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Journal of Occupational Medicine

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- THE TREATMENT OF SNAKEBITE ..... *A. C. Stimson and H. T. Engelhardt, M.D.*
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Vol. 2 No. 4
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Journal of Occupational Medicine

Official Publication of the Industrial Medical Association

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PART II—*Harry Levinson, Ph.D., Topeka, Kan.*

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NEWER CONCEPTS OF LOW BACK PAIN
M. Laurens Rowe, M.D., Rochester, N.Y.

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Materials for publication should be addressed to the Editor, 2057 Beechwood Boulevard, Pittsburgh 17. Articles will be considered for publication on condition that they are submitted solely to the *Journal of Occupational Medicine*.

The manuscript, including references, footnotes, and legends for illustrations, should be typed on one side of a standard-sized page of opaque paper, double-spaced, and with at least a one inch margin at each edge. One carbon copy should accompany the original typescript, and the author should retain a carbon copy for use in reading galley proofs.

The authors' names should be accompanied by the highest academic or medical degrees that they hold; letters indicating membership in honor or professional societies will not be published. Authors who are in the armed services should not use civilian degrees in connection with their names. The city of each author should be supplied, and an address should be provided at the end of the manuscript for the use of readers who may wish to communicate with the senior author. If academic, hospital, or industrial connections are given for one author, they must be given for all other authors who have such connections. The authors are responsible for obtaining permission from the proper authorities to use the names of institutions.

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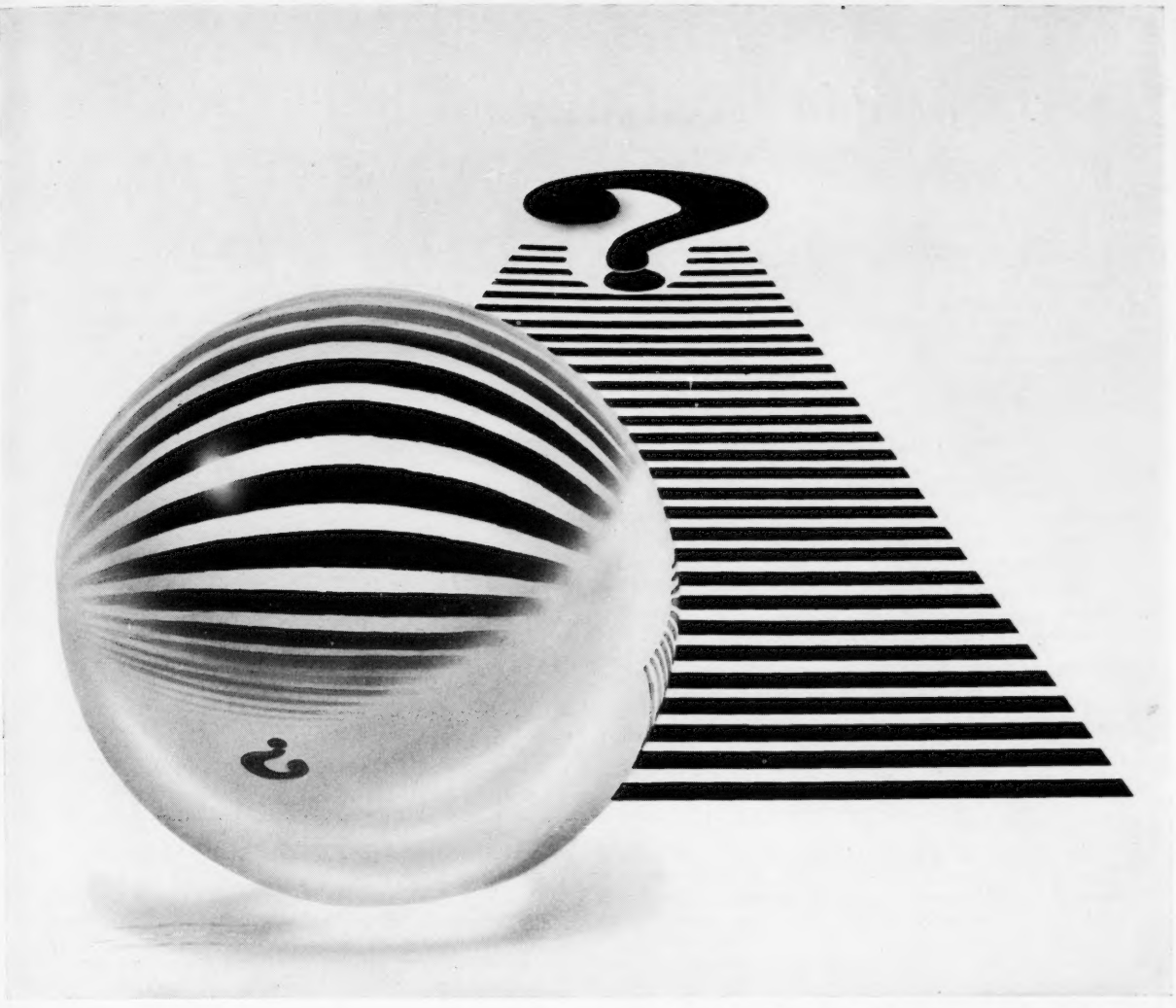

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References: 1. Dowling, H. F.: Postgrad. Med. *23*:594 (June) 1958. 2. Gimble, A. I.; Shea, J. G., and Katz, S.: Antibiotics Annual 1955-1956 New York, Medical Encyclopedia Inc., 1956, p. 676. 3. Long, P. H., in Kneeland, Y., Jr., and Wortis, S. B.: Bull. New York Acad. Med. *33*:552 (Aug.) 1957. 4. Rein, C. R.; Lewis, L. A., and Dick, L. A.: Antibiotic Med. & Clin. Ther. *4*:771 (Dec.) 1957. 5. Stone, M. L., and Mersheimer, W. L.: Antibiotics Annual 1955-1956, New York, Medical Encyclopedia Inc., 1956, p. 862. 6. Campbell, E. A.; Prigot, A., and Dorsey, G. M.: Antibiotic Med. & Clin. Ther. *4*:817 (Dec.) 1957. 7. Chamberlain, C.; Burros, H. M., and Borromeo, V.: Antibiotic Med. & Clin. Ther. *5*:521 (Aug.) 1958. 8. From, P., and Alli, J. H.: Antibiotic Med. & Clin. Ther. *5*:639 (Nov.) 1958.

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The Industrial Medical Association was organized in 1915 as the American Association of Industrial Physicians and Surgeons. Its first annual meeting was held the following year. In 1951 the present name was adopted.

The purposes of the organization as stated in the certificate of incorporation (not-for-profit) are:

"The object of this Association will be to foster the study and discussion of the problems peculiar to industrial medicine and surgery; to develop methods adapted to the conservation of health among workers in the industries; to promote a more general understanding of the purposes of the medical care for employees; and to unite into one organization members of the medical profession specializing in industrial medicine and surgery for their mutual advancement in the practice of their profession."

Presently, the membership totals nearly 4000 physicians in the United States, Canada, Latin America, and other parts of the world. Physicians "who have an interest in Industrial Medicine" may apply for membership; specialization in this field is not essential. Regionally, the Industrial Medical Association has 15 Component Societies in the United States. Most Association members carry their membership through one of the component groups.

The Journal of Occupational Medicine, owned and published by the Industrial Medical Association, is sent monthly to each member as one of the advantages of membership.

The annual business meeting and convention is held in the spring. Since 1945, the convention has been held concurrently with the meetings of four other organizations having related interests—American Industrial Hygiene Association, American Conference of Governmental Industrial Hygienists, American Association of Industrial Nurses, Inc., American Association of Industrial Dentists. This combined meeting is known as the Industrial Health Conference.

The Occupational Health Institute, Inc., is a nonprofit educational organization created by the Industrial Medical Association to promote the cause of health in industry. Since 1951, the Occupational Health Institute has conducted the program of evaluation, approval and certification of Medical Services in Industry, on the basis of standards established by the Standards Committee of the Industrial Medical Association. This program was conducted by the American College of Surgeons for many years until its transfer to the Association.

The Association's headquarters office serves as a focal point for the Association's activities, and through its coordination of information regarding industrial health problems encountered throughout the country, it offers invaluable assistance to members on a host of specific subjects.

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Association Activities

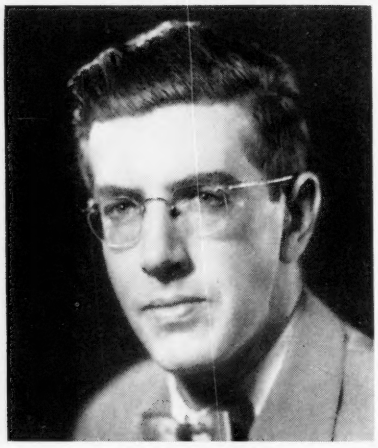
Officers and Directors Nominated; To Be Voted Upon at Annual Business Meeting

R. E. ECKARDT, M.D., President-Elect of the Industrial Medical Association, will automatically become President at the conclusion of the annual meeting in April. As provided in the By-Laws of the Association, nominations for the other offices have been made by the Board of Directors. These nominations automatically advance the First and Second Vice-Presidents in the line of succession to the presidency. Thus, GRADIE R. ROWNTREE, M.D., who is now First Vice-President, has been nominated for President-Elect; and H. GLENN GARDINER, M.D., who is now Second Vice-President, has been nominated for First Vice-President. LEONARD ARLING, M.D., was nominated for reelection as Secretary.

JOHN L. NORRIS, M.D., has been nominated for the office of Second Vice-President. DR. NORRIS, who is Associate Medical Director for Eastman Kodak Company, Rochester, New York, has been Treasurer of the Association since 1958. He joined IMA in 1939 and became a Fellow in 1945. He served on the Board of Directors from 1955 to 1957. He has been Chairman of the Finance Committee since 1956, and a member of the Journal Committee since last May. His medical degree was received from McGill University Medical School in 1931. He is a Fellow of the American Academy of Occupational Medicine and a Diplomate of the American Board of Preventive Medicine, certified in the specialty of Occupational Medicine, 1955.

JERMYN F. MCCAHAN, M.D., has been nominated for the office of Treasurer. DR. MCCAHAN is Medical Director for Western Electric Company, Chicago, Illinois. He has been a member of IMA since 1943

President-Elect and Nominees for Other Offices



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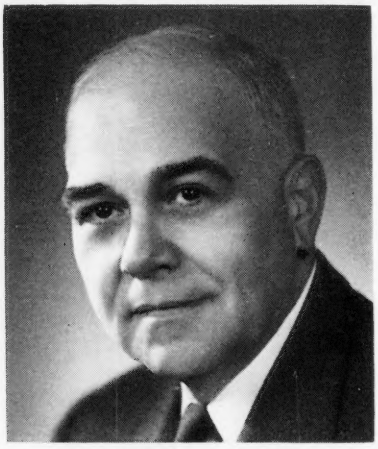

Gradie R. Rowntree, M.D.
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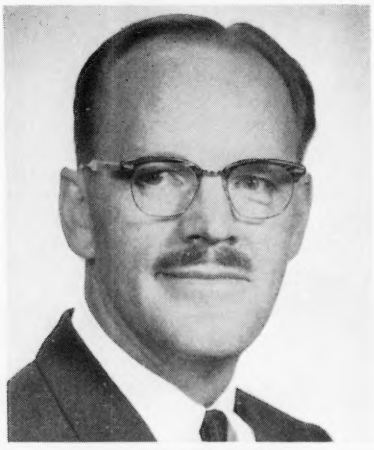
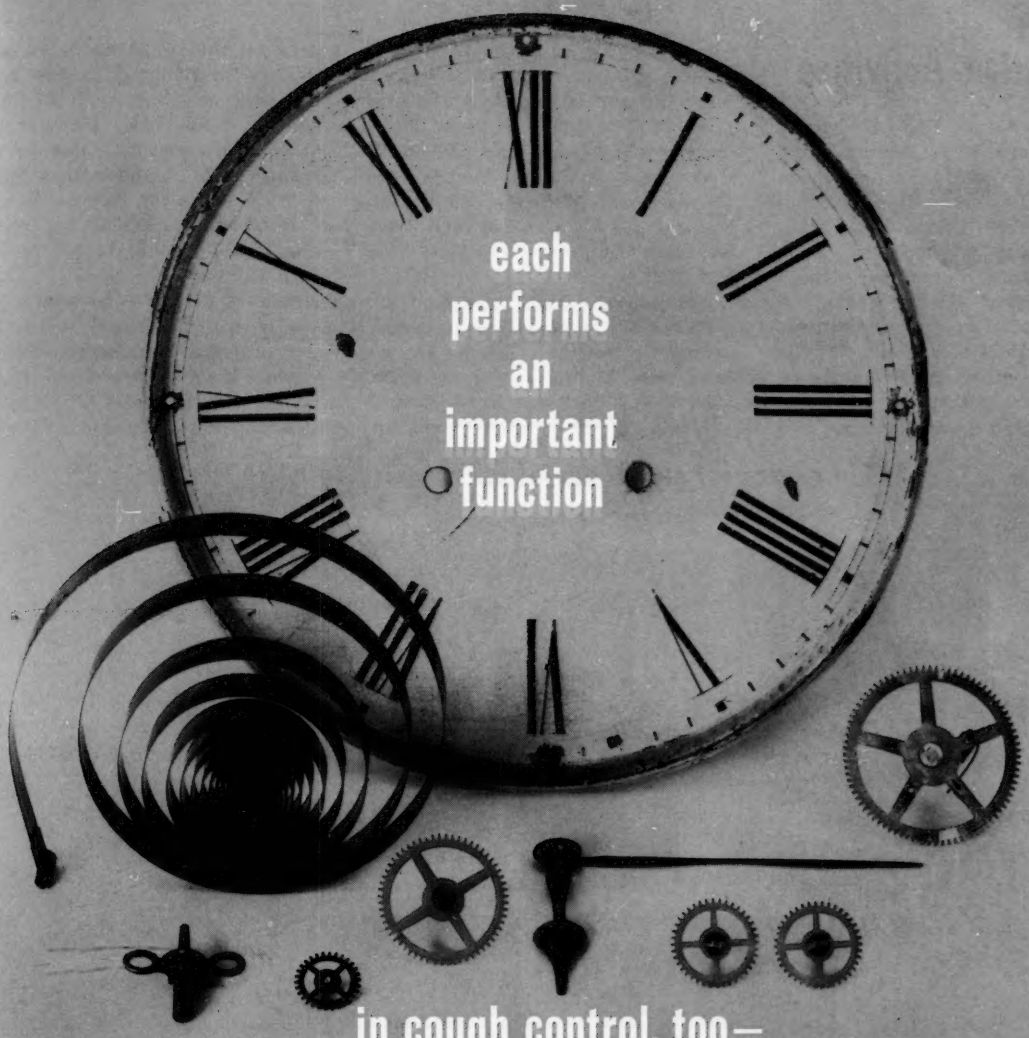
Nominee for Second Vice-President



Leonard Arling, M.D.
Nominee for Secretary

Jermyn F. McCahan, M.D.
Nominee for Treasurer



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Candidates for the Board of Directors are nominated by the Nominating Committee. Ten persons have been nominated for the term, 1960-1963. Five are to be elected.

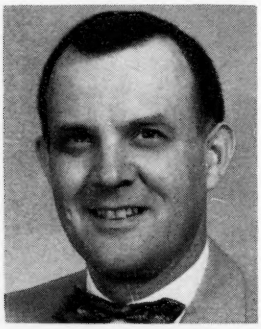
Names of all nominees will appear on the prepared ballot which will be distributed at the annual business meeting April 27 in Rochester, New York. Further nominations, however, may be made from the floor, and names may be written in on the ballot.

Brief biographical information about each candidate for directorship follows:

MERLE BUNDY—Assistant Medical Director, United States Steel Corporation, Pittsburgh, Pennsylvania; M.D. degree from Indiana University School of Medicine, 1942; Fellow, American Public

Nominees for the Board of Directors, 1960-1963

(Five to be Elected)



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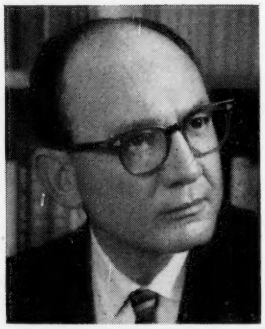

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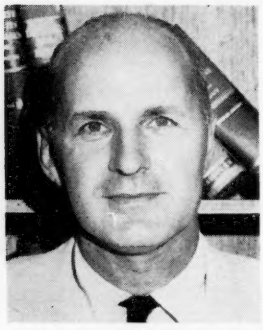

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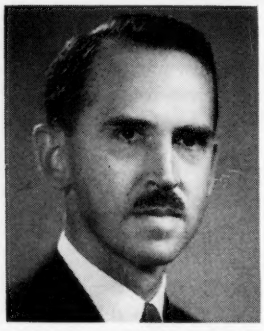

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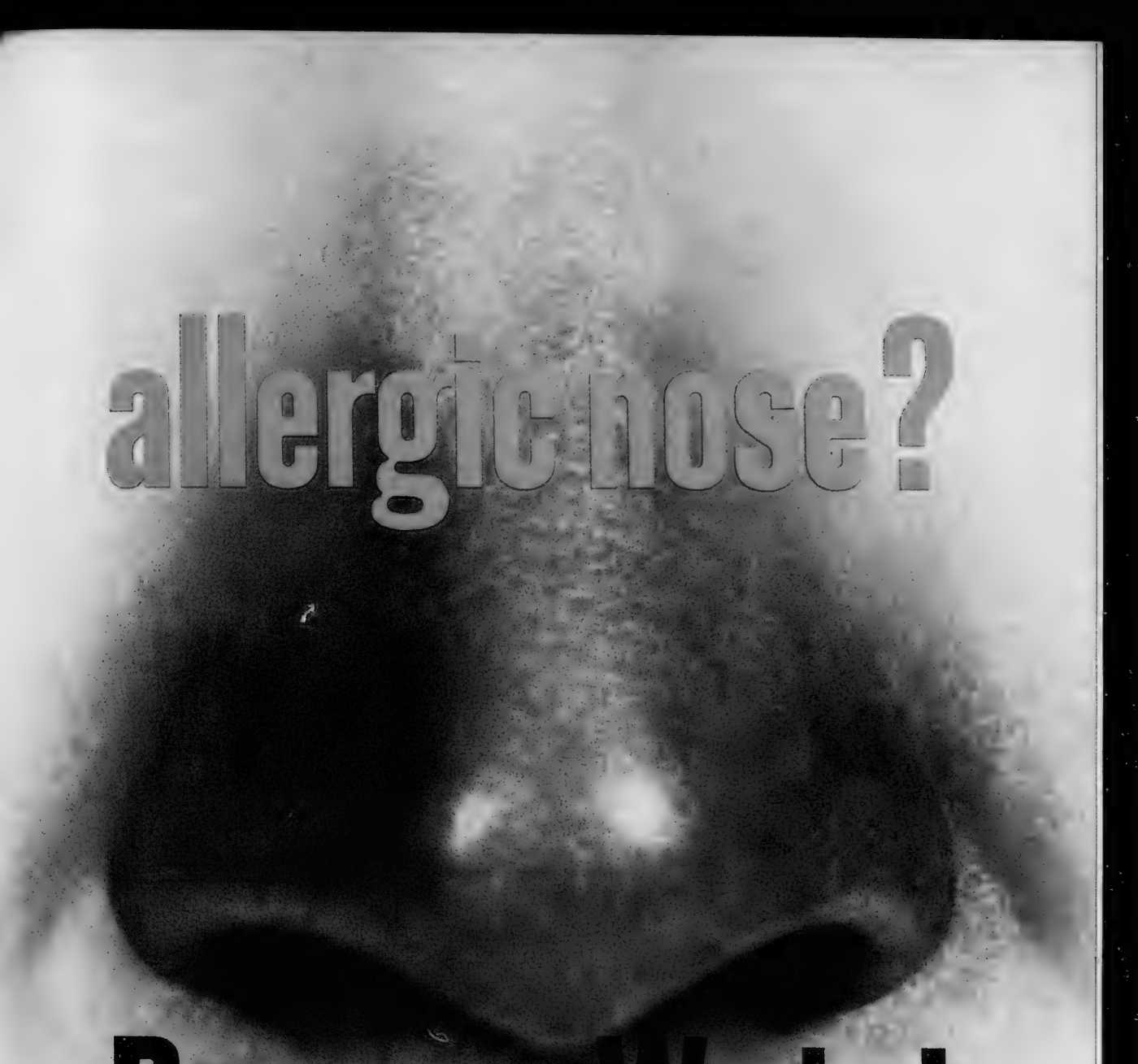

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Health Association; Fellow, American College of Preventive Medicine; Diplomate, American Board of Preventive Medicine, certified in Public Health, 1950, and Occupational Medicine, 1958. IMA activities: member, 1954; Fellow, 1959; District Counselor, 1955-1957; member, Public Relations Committee, 1957; member, By-Laws Committee, 1959 to present; Secretary-Treasurer, IMA of Pittsburgh, 1954 to present.

C. ANTHONY D'ALONZO—Assistant Medical Director, E. I. du Pont de Nemours & Company, Wilmington, Delaware; M.D. degree from the University of Virginia, 1937; Fellow, American College of Physicians; Fellow, American Academy of Occupational Medicine; Diplomate, American Board of Internal Medicine, 1952; Diplomate, American Board of Preventive Medicine, certified in Occupational Medicine, 1955. IMA activities: member, 1940; Fellow, 1948; Chairman, Cardiovascular Diseases Committee, 1958 to present.

J. S. FELTON—Professor of Occupational Health, Department of Preventive Medicine and Public Health, University of California School of Medicine, Los Angeles; M.D. degree from Stanford University, 1935; Fellow, American Academy of Occupational Medicine; Fellow, American Public Health Association; Diplomate, American Board of Preventive Medicine, certified in Occupational Medicine, 1955. IMA activities: member, 1944; Fellow, 1950; member, Committee on Education and Training, 1952-1957, and Chairman, 1957 to present; member, Committee on Problem Drinking, 1952-1954; member, Health Achievement in Industry Award Committee, 1959 to present.

MAC ROY GASQUE—Medical Director, Olin Mathieson Chemical Corporation, Pisgah Forest, North Carolina; M.D. degree from University of Virginia, 1944; Fellow, American Academy of Occupational Medicine; Fellow, American College of Preventive Medicine; Diplomate, American Board of Preventive Medicine, certified in Occupational Medicine, 1957. IMA activities: member, 1949; Fellow, 1954; District Counselor, 1953 and 1959; member, Committee on Industrial Medical Practice, 1959 to present.

DAVID H. GOLDSTEIN—Medical Director, *The New York Times*, and Professor of Industrial Medicine, New York University Bellevue Medical Center; M.D. degree from New York University College of Medicine, 1933; member, American Academy of Occupational Medicine; member, American Public Health Association; Diplomate, American Board of Internal Medicine, 1943, and American Board of Preventive Medicine, certified in Occupational Medicine, 1955. IMA activities: member, 1953; Fellow, 1956; member, Committee on Education and Training, 1956 to present; President, New York State Society of Industrial Medicine, 1958-1959.

DAVID D. HOLADAY—Area Physician, American Can Company, San Francisco, California; M.D. degree from University of Kansas, 1935; Diplomate, American Board of Preventive Medicine, certified in Occupational Medicine, 1955. IMA activities: member, 1950; Fellow, 1956; member, Membership Committee, 1959 to present; President, Western Industrial Medical Association, 1957-1958.

SEWARD E. MILLER—Director, Institute of Industrial Health, and Chairman, Department of Industrial Health, School of Public Health, University of Michigan, Ann Arbor; M.D. degree from University of Michigan, 1931; member, American Public Health Association, Association of Military Surgeons and American Society of Clinical Pathologists. IMA activities: member, 1951; Fellow, 1958; member, Committee on Standards for Motor Vehicle Driving, 1956 to present.

GORDON A. SINCLAIR—Assistant Medical Director, Imperial Oil, Ltd., Toronto, Ontario; M.D. degree from University of Toronto, 1926; Chairman, Industrial Section, Ontario Medical Association, 1952-1953; Chairman, Section on Industrial Medicine, Academy of Medicine, 1949-1950; Fellow, American Academy of Occupational Medicine; Diplomate, American Board of Preventive Medicine, certified in Occupational Medicine, 1955; Certified Specialist in Internal Medicine, Royal College of Physicians and Surgeons (Canada). IMA activities: member, 1947; Fellow, 1954.

RICHARD A. SUTTER—Director, Sutter Industrial Clinic, St. Louis, Missouri; M.D. degree from Washington University (St. Louis), 1935; member, American Industrial Hygiene Association; Fellow, American College of Preventive Medicine; member, American Association of Railway Surgeons; Diplomate, American Board of Preventive Medicine, certified in Occupational Medicine, 1956. IMA activities: member, 1946; Fellow, 1953; District Counselor, 1951-1952; Deputy General Chairman, Industrial Health Conference, 1957; member, Committee on Surgery of Trauma, 1953-1955; member, Health Achievement in Industry Award Committee, 1956-1958, Chairman, 1958 to present; Board of Governors, Central States Society of Industrial Medicine and Surgery, 1952-1955, and President, 1959-1960.

PAUL J. WHITAKER—Medical Director, Allis-Chalmers Manufacturing Company, Milwaukee, Wisconsin; M.D. degree from University of Pittsburgh, 1939; Fellow, American Academy of Occupational Medicine; Diplomate, American Board of Preventive Medicine, certified in Occupational Medicine, 1956. IMA activities: member, 1952; Fellow, 1956; Chairman of Scientific Exhibits, 1954 and 1958; member, Organization Committee, 1959 to present; member, National Safety Council Medical Advisory Committee, 1959 to present; President-Elect, Central States Society of Industrial Medicine and Surgery.

Occupational Medicine Examinations Scheduled

Examinations for specialty certification in occupational medicine will be conducted by the American Board of Preventive Medicine preceding the Industrial Health Conference in Rochester, N.Y. The written examinations will be given April 22 and 23 in the Manger Hotel. The oral examinations will be given on April 24.

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Kenacort is available in 1 mg., 2 mg., and 4 mg. scored white tablets.

References: 1. McGavack, T. H.: Clin. Med. *6*:997 (June 1959). 2. Plotz, C. M.: Paper on administration of corticosteroids in rheumatoid arthritis, presented at the 11th Annual Scientific Assembly of the New York Academy of General Practice, New York City, (October 20, 1959). 3. Williams, G. T: Southern Med. J. *52*:267-273 (March 1959).

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Association Activities

Technical Exhibits to Feature New Products and Equipment at Industrial Health Conference

The newest products and equipment applicable to the field of industrial health will be on display at the War Memorial Auditorium, Rochester, N.Y., April 26 through 28 during the 1960 Industrial Health Conference. The companies with Technical Exhibits scheduled to date include the following:

Exhibitor	Booth Number
Air Shields, Inc. Hatboro, Pa.	230
Alcon Laboratories, Inc. Fort Worth, Texas	317
A. S. Aloe Company St. Louis, Mo.	219-220
American Cancer Society New York, N.Y.	316
American Optical Company Southbridge, Mass.	322
American Sterilizer Company Erie, Pa.	129
The American Tobacco Company Richmond, Va.	234-235
Armour Pharmaceutical Company Kankakee, Ill.	203
Association of American Soap and Glycerine Producers, Inc. New York, N.Y.	301
Astra Pharmaceutical Products Worcester, Mass.	205
Ayerst Laboratories New York, N.Y.	217
Bausch & Lomb Optical Company Rochester, N.Y.	226-227
Beltone Hearing Aid Company Chicago, Ill.	215
Bristol-Myers Products New York, N.Y.	231
Buffington's, Inc. Worchester, Mass.	204
Cambridge Filter Corporation Syracuse, N.Y.	136
Carbisulphoil Company Dallas, Texas	229
Chattanooga Pharmacal Company, Inc. Chattanooga, Tenn.	11
Chloraseptic Corporation	212
Chrysler Corporation Detroit, Mich.	326-327-328-329-330
Ciba Pharmaceutical Products Summit, N.J.	210
Coca Cola Company Atlanta, Ga.	319-320
Dameron Enterprises, Inc. Louisville, Ky.	310
Davis Emergency Equipment Co. Newark, N.J.	222
Dome Chemicals, Inc. New York, N.Y.	110
Duke Laboratories, Inc. South Norwalk, Conn.	303
Eastman Kodak Company Rochester, N.Y.	232
Geigy Pharmaceuticals Yonkers, N.Y.	119
Gelman Instrument Company Chelsea, Mich.	2
Gideons International Chicago, Ill.	334
Globe Industries, Inc. Medical and Hospital Div. Dayton, Ohio	6
Great Books of the Western World Grand Rapids, Mich.	111
A. E. Halperin Company, Inc. Boston. Mass.	302
Hillman Pharmaceutical Co. Chicago, Ill.	216
Industrial Medical Association History Comm. Chicago, Ill.	313
Industrial Acoustics Company, Inc. New York, N.Y.	308
The Iso-Sol Company, Inc. Lindenhurst, N.Y.	335
Johnson & Johnson New Brunswick, N.J.	12
Karel First Aid Supply Co. Chicago, Ill.	202
Keystone View Company Meadville, Pa.	206
Licensed Beverage Industries, Inc. New York, N.Y.	312
Eli Lilly & Company Indianapolis, Ind.	228
Lixoil Laboratories Boston, Mass.	10
Maico Electronics, Inc. Minneapolis, Minn.	225
Medco Products Company Tulsa, Okla.	137
Merck Sharp & Dohme Philadelphia, Pa.	201
Milburn Company Detroit, Mich.	221
Mine Safety Appliances Company Pittsburgh, Pa.	120-121-122
Oxy Swig, Inc. San Francisco, Calif.	118
G. H. Packwood Manufacturing Co. St. Louis, Mo.	233
Paine Drug Co. Rochester, N.Y.	4
Pepsi-Cola Company New York, N.Y.	213-214
Pfizer Laboratories Brooklyn, N.Y.	218
Physicians Supply Corporation Rochester, N.Y.	314
Purdue Frederick Company New York, N.Y.	3
Relax-A-Tron Products, Inc. Flint, Mich.	5
R. J. Reynolds Tobacco Company Winston-Salem, N.C.	101
Ritter Company, Inc. Medical Hospital Div. Rochester, N.Y.	309
Sanborn Company Cambridge, Mass.	9
Schering Corporation Bloomfield, N.J.	336
Schieffelin & Company New York, N.Y.	211
Smith, Kline & French Labs. Philadelphia, Pa.	14
E. R. Squibb & Sons New York, N.Y.	337
Stephenson Corporation Red Bank, N.J.	321
Stiefel Laboratories, Inc. Oak Hill, N.Y.	13
Sugar Beet Products Company Saginaw, Mich.	208-209
Syntex Chemical Company, Inc. New York, N.Y.	224
Tailby-Nason Company, Inc. New York, N.Y.	113
Titmus Optical Company, Inc. Peterburg, Va.	109
Union Industrial Equipment Corp. White Plains, N.Y.	1
United States Safety Service Company Kansas City, Mo.	311
United States Tobacco Company New York, N.Y.	223
The Upjohn Company Kalamazoo, Mich.	207
Vitamins for Industry, Inc. Los Angeles, Calif.	8
Wallace Laboratories New Brunswick, N.J.	128
Warner-Chilcott Laboratories Morris Plains, N.J.	236
Whitehall Laboratories Div. of American Home Products Corp. New York, N.Y.	102
Willson Products Reading, Pa.	315
Winthrop Laboratories New York, N.Y.	318

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TINEA PEDIS (ATHLETE'S FOOT) AND
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typical response of tinea pedis to GRIFULVIN

- itching and burning relieved in **2 to 6 days**
- vesicles and scaly patches disappear completely; cultures and KOH scrapings usually become negative in **2 to 6 weeks**
- side effects are rare, mild and transitory

Average dose: 250 mg. q.i.d. Adjunctive treatment with topical keratolytic agents will aid in eradicating the fungi from the skin of the feet.

Supplied: 250 mg. scored tablets, colored aquamarine, imprinted McNEIL, bottles of 16 and 100.

Blank, H., Smith, J. G., Jr., Roth, F. J., Jr., and Zaias, N.: J.A.M.A. 171:2168 (Dec. 19) 1959.
Photographs courtesy of Harvey Blank, M.D., Miami, Florida

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Association Activities

Actions of the Board of Directors at the Williamsburg Meeting in February

The Board of Directors of the Industrial Medical Association met at the Williamsburg Inn, Williamsburg, Virginia, on February 10, 1960. Following is an abstract of the minutes of the meeting:

Due to the unavoidable absence of President D. JOHN LAUER, M.D., who was out of the country at the time, the meeting was called to order by the President-Elect, ROBERT E. ECKARDT, M.D. The Board took the following actions:

Nominated officers for 1960-1961 as follows: GRADIE R. ROWNTREE, M.D., President-Elect; H. GLENN GARDINER, M.D., First Vice-President; JOHN L. NORRIS, M.D., Second Vice-President; LEONARD ARLING, M.D., Secretary; J. F. MCCAHAN, M.D., Treasurer.

Nominated for Directors for 1960-1963 (five to be elected): MERLE BUNDY, M.D., Pittsburgh, Pa.; C. A. D'ALONZO, M.D., Wilmington, Del.; JEAN S. FELTON, M.D., Los Angeles, Calif.; MAC ROY GASQUE, M.D., Pisgah Forest, N. C.; DAVID H. GOLDSTEIN, M.D., New York, N. Y.; DAVID D. HOLADAY, M.D., San Francisco, Calif.; SEWARD F. MILLER, M.D., Ann Arbor, Mich.; GORDON A. SINCLAIR, M.D., Toronto, Ont.; RICHARD A. SUTTER, M.D., St. Louis, Mo.; PAUL J. WHITAKER, M.D., Milwaukee, Wis.

Received report from E. S. JONES, M.D., Chairman of the Occupational Health Institute, that even though the Council on Industrial Health of the American Medical Association had approved and recommended the organization of a joint IMA-AMA Commission on Accreditation of Occupational Health Programs, the AMA Board of Trustees voted against such organization. Agreed to cooperate fully with DR. JONES in his efforts to interest an outstanding industrialist in accepting the chairmanship of OHI and promoting fuller activities of the OHI.

Approved Fellowship status for 41 active members on receipt of a report from the Board of Examiners for Fellowship.

Accepted "Criteria Governing the Establishment of Component Societies" developed by the By-Laws Committee.

Discussed forthcoming Industrial Health Conference and IMA annual meeting planning. Voted that the Health Achievement in Industry award and the Knudsen award be presented at the banquet, and the Meritorious Service award and Merit in Authorship award be given at the annual business meeting.

Received notice dated February 2, from CHARLES D. YAFFE, Secretary-Treasurer of the American Conference of Governmental Industrial Hygienists, to the effect that "the ACGIH membership has voted not to participate in the Industrial Health Conference in 1961. No definite decisions beyond 1961 have been made."

Approved report that the 1961 Industrial Health Conference has been cancelled out of Dallas and is to be held in the Palmer House, Chicago.

Accepted suggested plan for reorganizing the format of IMA preliminary sessions at the Industrial Health Conference: Joint Meeting of officers and directors, component society officers, district counselors, committee chairmen, nominees for directors, newly appointed counselors, and newly appointed committee chairmen, to be held on Sunday prior to annual meeting. Officers responsible for various committees were requested to notify chairmen of the March 1 deadline for receipt of reports, so that they might be duplicated before the annual meeting.

Voted that reports of Board of Directors actions constitute official notice when reported in the JOURNAL OF OCCUPATIONAL MEDICINE.

Approved budget of $7,000 for 1960 expenses of the JOURNAL OF OCCUPATIONAL MEDICINE Editorial Office.

Received report of Treasurer, JOHN D. NORRIS, M.D., for the calendar year 1959: Cash in bank on January 1, 1959, $28,007.00; on January 1, 1960, $28,115.00; with outstanding checks amounting to approximately $3,000.00. Membership increase during 1959: 243. The estimate for the fiscal year ending August 31, 1960, is that expenses will be about equal to income if a membership directory is not published. It was pointed out that the financial reserves are considerably less than those considered desirable for good association management, which requires a reserve at least equal to one year's operating expenses, and preferably equal to two. This matter to be taken under consideration by the Finance Committee.

Voted that the publication of a membership roster be tabled for the present. GRADIE R. ROWNTREE, M.D., and LOGAN T. ROBERTSON, M.D., were asked to study details of publication cost and report at the next Board meeting.

Deferred membership in National Health Council.

Considered the recommendation of the Committee on Industrial Medical Practice relative to a survey on the scope and functions of current occupational health programs. Project to be studied further as to its scope, timeliness, and other problems involved.

Received progress report from the History Committee on the publication of the history of the IMA. The Wayne State University Press has agreed to undertake this publi-

15th Component Society

The Kentucky Industrial Medical Association has become the 15th component society of the IMA. The petition of the Kentucky group was accepted by the IMA Board of Directors on February 10. The organization was formed last September by a group of 37 Kentucky members of IMA. Officers and directors are: GRADIE R. ROWNTREE, M.D., President; EUGENE H. KREMER, M.D., President-Elect; CHARLES F. MARTIN, M.D., Vice-President; DONALD B. THURBER, M.D., Secretary-Treasurer; ASA BARNES, M.D., director, one-year term; RICHARD E. DOUGHTY, M.D., director, two-year term; WALTER L. O'NAN, M.D., director, three-year term.

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Tessalon, a nonnarcotic, is 2½ times as effective as codeine.* Tessalon acts both at the sensory receptors in the chest *and* the cough centers of the medulla. Furthermore, it controls cough frequency without interfering with productivity or expectoration; sputum is usually thinner, easier to raise. Tessalon acts within 15 or 20 minutes, controls cough for 3 to 8 hours. There are no major side effects. Whether for acute or chronic cough, whether for short- or long-term therapy, Tessalon has a remarkable margin of safety. Perles insure built-in, precise dosage—no sugar or sodium to interfere with diet, no problem of nausea. Tessalon perles are easy to swallow, easy to carry in pocket or purse.

SUPPLIED: Tessalon *Perles*, 100 mg. (yellow); bottles of 100. Tessalon *Pediatric Perles* (for children under 10), 50 mg. (red); bottles of 100. *Also available* (for use when oral administration of Tessalon is precluded): *Ampuls*, 1 ml. (5 mg.); cartons of 5.

* Shane, S. J., Krzyski, T. K., and Copp, S. E.: Canad. M.A.J. 77:600 (Sept. 15) 1957.

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cation on a profit sharing basis. The volume which will be entitled, "Occupational Health in America," is scheduled for publication in 1960. The Board agreed to provide promotional assistance in the pages of the JOURNAL and at the Industrial Health Conference to help insure the success of this publication.

Tabled report of 1958-1959 Workmen's Compensation Committee.

Approved report of Radiation Committee for presentation at Rochester and for publication in the JOURNAL.

Received report that the Small Plants Services Committee plans to work with a newly formed similar committee of the American Academy of General Practice. The exhibit of the IMA committee will be shown at the AAGP meeting in Philadelphia and will be staffed by local members.

Received report of Committee on Standards to effect that the medical services of four companies were approved for certification, one was deferred and six survey reports under study.

Voted to accept invitation to serve on permanent Council on Medical Television, Institute for Advancement of Medical Communications. LEON WARSHAW, M.D., appointed as IMA delegate.

Accepted petition of the Kentucky Industrial Medical Association to become a component society.

Report of the History Committee

The History Committee of the IMA has just completed a history of the Association. It is called "Occupational Health in America," and carries the subtitle, "The Evolution of the Industrial Medical Association." After almost 20 years of planning, preparation, and production, the book is soon going to press. Just when the idea of such a history first suggested itself—and to whom—no one knows, but available records show that a history of some sort was officially discussed by the American Association of Industrial Physicians and Surgeons, forerunner of the IMA, in the early 1940's . . . and older members had talked about the project among themselves long before then.

The members urging immediate action on the plan included several from the little group that had led the way to a new era in American occupational medicine during the founding and formative years of the Association. Uppermost in their minds was the thought that unless a detailed account of that period were preserved in printed form, the personalities, ideas, and events of the time would fade from memory or remain buried in dusty files and scattered periodicals seldom available for ready reference. Thus, material of widespread interest and historical significance would be lost.

In 1942, a "committee for the purpose of preparing a history" was established. The immediate task was to assemble all pertinent data on health care in industry in the first two or three decades of the twentieth century. Ironically, detailed and reliable information on that colorful and exciting formative period in the story of industrial medicine proved to be less accessible than suitable background material from far older times. Records were scanty, and scattered; important activities had to be constructed, in many instances, from the memory of participants.

This must not be taken to mean that there was a dearth of writings in the field. The period from 1910 to 1940 produced a rapidly growing volume of significant literature relating to occupational health, but by far the greater part of it was naturally devoted to the medical and scientific aspects of industrial practice. Missing in this body of professional books and papers was a thread of connected narrative tracing the evolution of present-day occupational medicine against a background of social, economic and technological change.

In a broad sense, preparation of a history that would bridge this important gap was the primary aim of the Committee. And the work of gathering and correlating relevant material went on through the 1940's and into the 1950's as rapidly as the time of busy professional men permitted.

During the entire period there were few changes in the personnel of the History Committee, and few deviations from original policies. From 1943 to 1950 the project was under the direction of Committee Chairman DR. LOYAL A. SHOUDY, DR. OTTO P. GEIER, and DR. ALFRED H. WHITTAKER. Other members were DRS. FRANK L. RECTOR, C. F. N. SCHRAM, and C. O. SAPPINGTON. DR. WHITTAKER was appointed chairman in 1947.

Later, the Committee was augmented by the appointment of DRS. WILLIAM A. SAWYER (Eastman Kodak Company), JAMES B. MCCONNAUGHY (Aluminum Company of America), and H. G. MURRAY (Dennison Manufacturing Company). In the fall of 1951, the last major revision of personnel was made by the appointment of the following members:

DR. ROBERT T. LEGGE, educator, bibliophile, and historian; one of the founders' group of the Industrial Medical Association, with more than 50 years of active service in occupational medicine.

DR. MCIVER WOODY, a former professor of surgery, with an avid interest in medical history; president of the Association in 1939-1940, and for many years medical director of the Standard Oil Company (New Jersey).

DR. JAMES I. ROBERTS, a medical consultant for New England public utilities, with experience in the publishing field; a District Counselor of the Industrial Medical Association, and chairman of the Committee on Medical Care (1954-1955).

This volume, then, has taken shape as an account of the men, the events, and the times responsible for the emergence of occupational medicine from its formative stage, and the application of its protective and preventive techniques in a major area of our society—the environment in which men work and earn their livelihood.

—A. H. WHITTAKER, M.D., Chairman; ROBERT T. LEGGE, M.D.; MCIVER WOODY, M.D.; JAMES I. ROBERTS, M.D.

Journal of Occupational Medicine

A Company Program for Problem Drinking

TEN YEARS' FOLLOW-UP

S. Charles Franco, M.D., New York, N.Y.

INDUSTRY has become increasingly aware of the need to deal constructively with the employee who has a drinking problem. Experience has shown that the forthright recognition of problem drinking coupled with a program for rehabilitation has proved worth while. Also, it has been adequately demonstrated that the early recognition of a drinking problem offers the best opportunity for successful rehabilitation.

The Consolidated Edison Program

In December, 1947, the Consolidated Edison Company of New York, Inc., officially recognized chronic alcoholism as a medical condition and adopted a company procedure on alcoholism.[1] This decision was based on a policy that we would meet the problem openly instead of perpetuating the outworn pretense that it did not exist. The analysis of follow-up results has been recognized to be a long-term affair. To allow for proper critical evaluation, it was decided to use December, 1957, as the cut-off point for this 10-year study so that the minimal period of follow-up would be at least one year.

In the period 1948-1957, at Consolidated Edison Company, with a stable employee population, we recognized nearly 400 cases of a drinking problem. Our experience leads us to place the annual incidence of new cases at 1½ to 2 cases per 1,000 employees per year. We have averaged 40 new cases each year in our group of approximately 25,000 employees. Of course, not all cases are identified, even under a well-organized program, and we estimate that the real incidence in industry is approximately 2% to 3% of employees.

Presented at the 44th Annual Meeting of the Industrial Medical Association, Chicago, April 30, 1959.

Dr. Franco is Executive Medical Director, Consolidated Edison Company of New York, Inc.

Operation of the Company Procedure

The basis of the Consolidated Edison Company procedure on alcoholism is the use of a firm, judicious probation policy which, in effect, puts job security in jeopardy and motivates the employee to do something about his problem. This has been coupled with an adequate medical facility of rehabilitation.

The steps in the operation of the procedure on alcoholism are summarized as follows:

1. *Recognition*—The foreman and supervisor have the responsibility for the recognition of the early signs of the uncontrolled use of alcohol among men working under them. It is not the responsibility of the medical department to discover alcoholics, but it does play a role in the early recognition of a drinking problem masquerading as a medical illness.

When any department of the company is confronted with a behavior problem arising from repeated alcoholic excess, the employee is given a preliminary warning by his immediate supervisor. When there is a recurrence of the drinking situation, the employee is immediately interviewed by his supervisor and is placed on probation. The department head notifies the personnel director and furnishes him with a written report of all the circumstances to be forwarded to the medical department. Our experience indicates that a complete and detailed

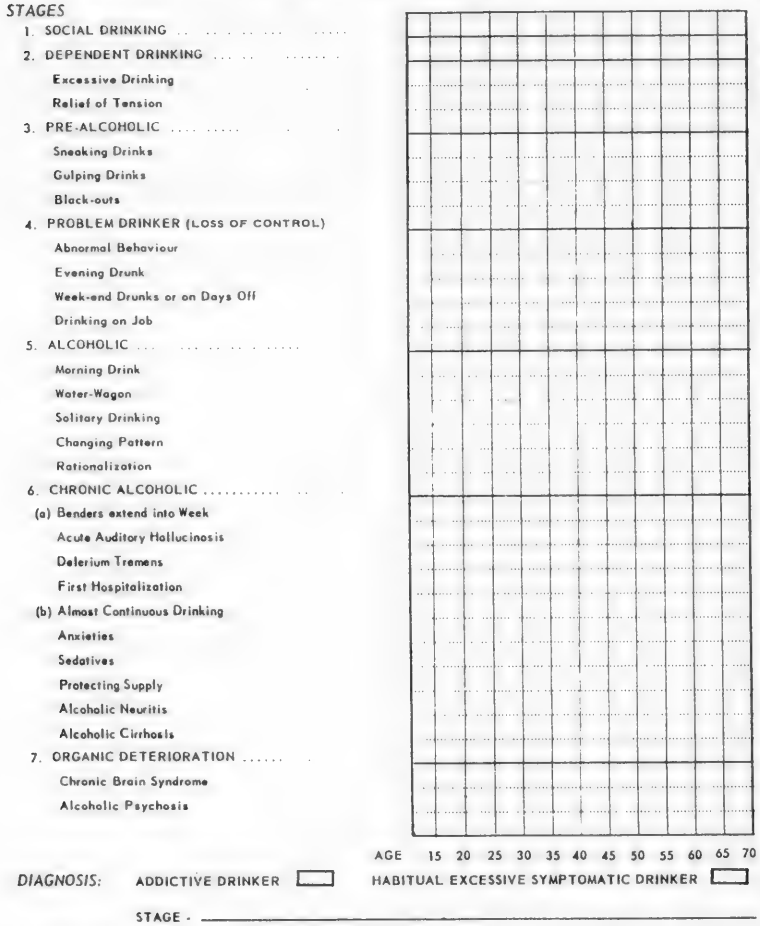
MEDICAL DEPARTMENT
CONSOLIDATED EDISON COMPANY OF NEW YORK, INC.

NAME _____ DATE _____

EMPLOYEE NUMBER _____ DEPT. _____

REFERRAL _____ AGE _____ SERVICE _____

* PROFILE OF ALCOHOLISM



*Clinical adaptation of "Phases in the Drinking History of Alcoholics" by E. M. Jellinek, Sc.D. and "Clinical Orientation to Alcoholism" by R. G. Bell, M.D.

Figure 1

report giving the entire story is a tremendous assistance to the physician in making his evaluation of the case. The employee in his interview with his immediate supervisor is advised that information he conveys to the examining physician may not be considered as a privileged communication.

A training program for supervisors and foremen is in continuous operation to instruct this group in the understanding of the objectives of the company program and to assist them in recognizing the early signs of problem drinking. The following are said to be indicative of the early stages of a drinking problem and often characteristic of the hidden alcoholic.

(*a*) *Absenteeism*—Absence on Monday morning and frequent occurrences of leaving early on Friday afternoon are noted. This will also include "no report" absences which are not covered by any medical certificate, as well as unexplained disappearance from assigned work.

(*b*) *Accidents*—Experience indicates that on-duty accidents are relatively rare in alcoholics, but they do frequent off-duty accidents, particularly with assault as a factor.

(*c*) *Personality*—Changes may become apparent in a previously good worker.

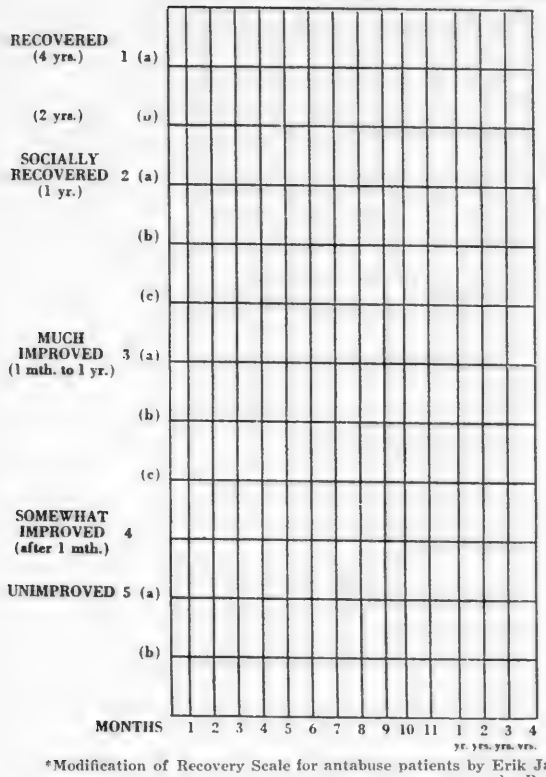
(*d*) *Efficiency*—There is reduction in job efficiency with operating errors and incompleted work.

2. REHABILITATION — The medical department has the responsibility for the diagnosis of alcoholism (Fig. 1) and for the supervision of follow-up for rehabilitation (Fig. 2).[2] The employee is offered the opportunity for rehabilitation at the Consultation Clinic for Alcoholism at the University Hospital. On the other hand, he may choose to report to Alcoholics Anonymous, or place himself under the care of his own physician, or even refuse to follow any recommended treatment. It is interesting to note that since the clinic facilities have become available, approximately 75% of the employees with a drinking problem have accepted referral for treatment.

It has been our experience that treatment itself is best carried on outside the company setting. However, some companies have reported good results with in-plant treatment. The Dupont and the Eastman Kodak companies have been successful in the use of Alcoholics Anonymous,* and other companies, such as Consolidated Edison, have had their best results with special medical clinics. At the con-

*Personal communications from George H. Gehrmann, M.D., (E. I. du Pont de Nemours & Co.) and John L. Norris, M.D. (Eastman Kodak Co.).

*RECOVERY SCALE FOR DEPENDENT DRINKING


RECOVERED (4 yrs.) 1 (a)
(2 yrs.) (b)
SOCIALLY RECOVERED (1 yr.) 2 (a)
(b)
(c)
MUCH IMPROVED (1 mth. to 1 yr.) 3 (a)
(b)
(c)
SOMEWHAT IMPROVED (after 1 mth.) 4
UNIMPROVED 5 (a)
(b)
MONTHS 1 2 3 4 5 6 7 8 9 10 11 1 2 3 4
yr. yrs. yrs. yrs.

1. *Recovered*
   (a) (This classification applicable only after 4 years uninterrupted total abstinence) All dependence on alcohol gone and replaced by a new system of satisfactory, mature adjustments and values.
   (b) (This classification applicable only after 2 years uninterrupted total abstinence) Dependence on alcohol appears to be gone and to be replaced by a progressively improving new system of satisfactory, mature adjustments and values.
2. *Socially Recovered*—(This classification only applicable one year after completion of inital treatment)
   (a) Has illness under control. No relapses. Patient aware of a progressive improvement in capacity to adjust to life without former recourse to alcohol.
   (b) No relapses. Retains good motivation but still has significant psychological, social or metabolic problems. Requires further assistance.
   (c) Occasional relapses not severe enough to impair new social status (home, employment, community, etc.). Retains good motivation but definitely requires further assistance.
3. *Much Improved*—(This classification applicable during first year and after first month following completion of initial treatment)
   (a) Has illness under control. No relapses. Patient aware of a progressive improvement in capacity to adjust to life without former recourse to alcohol.
   (b) No relapses. Retains good motivation but has significant psychological, social or metabolic problems. Likely will require further assistance before he can improve.
   (c) Occasional relapses not severe enough to impair new social status (home, employment, community, etc.). Retains good motivation but definitely requires further assistance.
4. *Somewhat Improved*—(This classification applicable after first month following completion of initial treatment) Relapses occurring severe enough to jeopardize social status (home and work) but improvement in motivation and effort over pretreatment period quite evident. Must have retention of sobriety for at least one month. Requires further assistance at once.
5. *Unimproved*—(This classification applicable at any time)
   (a) No noticeable change. Still considered candidate for treatment.
   (b) Considered to be a hopeless candidate for any type of treatment at present time.

*Modification of Recovery Scale for antabuse patients by Erik Jacobsen, M.D., Copenhagen, from "Clinical Orientation to Alcoholism" by R. G. Bell, M.D.

Figure 2

sultation clinic, the treatment consists essentially of a type of group therapy, and attempts are made to obtain an understanding of the alcoholic in regard to his immediate problems with drinking. Efforts at deep type of psychotherapy are usually avoided. It has been our experience that rehabilitation can be carried out on an ambulatory basis and only rarely is hospitalization or institutional care required. However, it is most important that the confidential nature of the doctor-patient relationship at the consultation clinic be maintained and no reports are required from the clinic.

3. DISPOSITION—If the drinking offense recurs, the employee is immediately suspended by his department and a Disability Panel for adjudication of his case is convened. At the Panel, after all information has been presented and evaluated, the department renders its decision. While alcoholism is treated as a medical condition, it gets no disproportionate share of attention. Means are provided to discipline and even discharge the offender whose problem does not seem to be related to an underlying psychological or medical situation.

When recurrences indicate an inability to cope with chronic problem drinking or there is underlying disease resulting from chronic alcoholism and the prognosis is poor, the employee is recommended for disability retirement. If the prognosis is good for recovery and the employee evidences every intention of facing his problem, a relapse of the drinking situation is not the occasion to deny him another chance at rehabilitation. As a matter of Company policy, sick pay is not approved for episodes of acute intoxication.

Alcoholism in Industry

From an industrial point of view, an employee is considered to be an alcoholic when repeated or continued overindulgence in alcoholic beverages interferes with the efficient performance of his assigned duties. This limits the recognition of abnormal drinking to the area where it affects work performance, causes abnormal behavior on the job, or results in absenteeism. The term "problem drinking" is often used synonymously with alcoholism in industry. Technically speaking it is considered an early stage of the development of chronic alcoholism.

We recognize two major types of alcoholics[3]: (1) the habitual excessive symptomatic drinker (the nonaddictive alcoholic), whose condition is primarily symptomatic of a social situation,

and (2) the alcohol addict whose drinking has the element of compulsion and whose condition appears to be based on an underlying psychological abnormality. It is our experience that the nonaddictive alcoholic represents between 80% and 85% of the problem drinkers that we recognize in industry. We also realize that, while some employees with a drinking problem will reveal examples of various diagnostic psychiatric conditions, therapy directed to the abnormal drinking behavior often appears to benefit the underlying psychiatric disturbance.

Alcoholism is a progressive disease, and a period from 2 to 15 years may pass from the development of the early stage of problem drinking to the establishment of the chronic phases of alcoholism with organic deterioration. The alcohol addict travels this road fast. In the prealcoholic phase, the alcoholic addict experiences blackouts after a relatively moderate alcohol intake. The nonaddictive alcoholic may also experience blackouts, but infrequently and only after marked intoxication.

The problem drinker can be defined as the early alcoholic. Often the results of his abnormal drinking are masked by being attributed to other causes. Although he believes that he can control his drinking, it has reached such proportions that it is beginning to affect his relationship with his family, his friends, and his employer. As his condition progresses, the problem drinker finally drinks on the job, even in situations where operating rules strictly prohibit it. The problem drinker is considered to have entered the alcoholic stage when he needs the morning drink to face the next day. At this point, he has lost control of his drinking situation and he begins to rationalize his behavior with a sequence of alcoholic alibis, solitary drinking, a change in the pattern of drinking, and even periods of abstinence under social pressures.

The chronic alcoholic may be rather dramatically identified by the onset of prolonged periods of intoxication, often referred to as benders. However, it is more common for chronic alcoholism to develop insidiously and for the effects of chronic overindulgence to remain hidden for a number of years. Continued excessive drinking can go on to produce physical effects on the body and can often result in the individual's developing one or more of the complicating diseases of chronic alcoholism, such as cirrhosis of the liver, peripheral neuritis, general malnutrition, and even beri-beri heart disease. It is unfortunate that a certain number of these persons are "hidden" alcoholics. The final stage of organic deterioration may be manifested by such diseases as the chronic brain

TABLE I—ALCOHOLISM PROGRAM: 1948-1957
GENERAL STATISTICS

Years	Total Cases
1948-1951	145
1952	53
1953	61
1954	33
1955	36
1956	40
1957	30
	398 (4 females)

Type of Referral	
Company procedure	358
Medical	31
Voluntary	9
	398

Classification	1948-1951	1952-1955	1956-1957
Early alcoholic	20%	48%	55%
Chronic alcoholic	60%	44%	45%
Advanced alcoholic	20%	8%	—

snydrome or an actual psychosis. At this stage, the individual may be known as a psychopathic alcoholic.

It is evident that many of the alcoholics in industry, even in companies with an alcoholism program, masquerade under other categories. Some are covered by certificates provided by their family physicians. Others are hidden by industry itself for reasons of years of faithful service or the idea that there is no adequate treatment. This has given rise to the erroneous concept in some quarters that alcoholism may not be considered a problem in industry. Medical experience discloses that unless there is actually a company program in operation, management cannot have any accurate knowledge of the extent of the problem.

Consolidated Edison Experience

Our experience under the procedure on alcoholism can be divided into two periods: The first from January, 1948, to December, 1951, and the second since January, 1952, when the facilities of the Consultation Clinic for Alcoholism at the University Hospital became operative. The major shortcoming of the program in the first period was that medical facilities for adequate rehabilitation were not available. Some persons derived great benefit from the AA Program but these were relatively few. Convinced by the lessons of its own internal program of the need of a special treatment center, top management at Consolidated Edison in December, 1951, volunteered to underwrite the cost of launching the Consultation Clinic for Alcoholism at the University Hospital of

TABLE II—ALCOHOLISM PROGRAM: 1952-1957
METHOD OF TREATMENT
(Reviewed March 1959)

	Total No.	Maintained Jobs No.	Maintained Jobs %	Lost Jobs No.
1. Referred to clinic	190	114	60	76
a. Continued treatment	128	87	60	41
b. Discontinued treatment or terminated in 6 months	62	27	43	35
2. Refused clinic	47	27	57	20
3. Not referred to clinic	16	5	31	11
	253	146	57	107

the New York University-Bellevue Center. The clinic was formally opened on Feb. 4, 1952, and the Standard Oil Company of New Jersey joined in the use and support of the clinic facilities in October of that year. Since then, approximately 20 companies have taken the opportunity to refer problem drinking cases. The results of treatment at the consultation clinic have been a vast improvement over our previous experience.

An analysis of the general statistics (Table I) reveals that the majority of the cases were recognized under the company procedure on alcoholism, and this reflects on the inability of purely medical or voluntary measures to face up to problem drinking in industry.

A review of the age and service of this group indicates that the high point comes between 46 and 54 years of age, when the patient has had over 20 years service. These employees obviously are a relatively stable group. The long years of employment have served as a lever in developing proper motivation toward treatment, because the probation process under the alcoholism procedure places job security in jeopardy.

Classification of the types of alcoholics reveals that with the experience gained under the procedure on alcoholism an increasing number of cases are being recognized in early stages.

A breakdown of the follow-up experience based on the method of treatment for the period 1952 through 1957, when the facilities of the consultation clinic for alcoholism were available, is shown in Table II. Out of a total of 253 cases, 190, or 75%, were referred to the clinic. Forty-seven employees refused the treatment at the clinic and were followed only in the medical department. These were largely the early-type alcoholic and problem drinkers. In some of these cases, the personal crisis precipitated by the probation procedure may motivate them to stop or control their drinking. Sixteen employees were not referred to the Clinic; seven were nonalcoholic, three cases presented predominant psychiatric disease, and six others were advanced alcoholics with marked organic deterioration.

A closer analysis of the results reveals that, of the employees who were able to continue treatment at the consultation clinic for at least one year, 60% maintained their jobs. Even more significant is that in this group who continued treatment more than 60% are considered socially recovered and 30% are much improved. By contrast, among the 57% who maintained their jobs in the group of 47 cases in which treatment at the consultation clinic was refused, only 25% have made real progress toward social recovery.

A review of the method of termination in the 107 employees who lost their jobs reveals that it was rather evenly divided between disability annuities (45%) and separation allowances (40%) which are limited as to duration. Sixty-five per cent of those who lost their jobs did so within the first year of follow-up. After the fourth year, relapses with failure and loss of jobs are rare. To illustrate that the alcoholic procedure was not intended as a reward for misbehavior about 10% of the drinkers were discharged and 5% resigned their jobs when they had a recurrence of their drinking behavior.

Analysis of the follow-up results of rehabilitation (Table III) has been shown to be a long-term affair. It is obvious that with longer follow-up the percentage of rehabilitated patients falls off. Also, the figures in the original group from 1948 to 1951, who did not have any special type of treatment, were adversely affected by the development of the diseases of chronic alcoholism as well as pulmonary tuberculosis, which conditions we are not encountering as frequently in the medically treated group.

An interesting feature of this study was that the evaluation by the operating departments of the progress of the employees under the proce-

TABLE III—ALCOHOLISM PROGRAM: 1948-1957
TEN-YEAR FOLLOW-UP
(Reviewed March 1959)

Years	Cases	Maintained Jobs No.	Maintained Jobs %
1948-1951	145	59	40
	Reviewed in 1956, 42%		
	Reviewed in 1953, 53%		
1952-1955	183	98	53
	Reviewed in 1956, 60%		
1956-1957	70	48	68
Total	398	205	51
1958	42	31	76

TABLE IV—ALCOHOLISM PROGRAM: 1948-1957

Cause of Death	Alcoholism Recovery Scale
1. Chronic alcoholism and fractured skull	Poor
2. Chronic alcoholism	Poor
3. Hepatic failure	No treatment
4. Hepatic failure	Poor
5. Hepatic failure	Poor
6. Pneumonia	Poor
7. Coronary thrombosis (female)	Poor
8. Cause unknown	Poor
9. Cancer of stomach	Much improved
10. Cancer of stomach	Recovered
11. Cancer of pancreas	Recovered
12. Cancer of lung	Recovered
13. Acute pancreatitis	Much improved
14. Ruptured peptic ulcer	Improved
15. Mesenteric thrombosis	Recovered
16. Coronary thrombosis	Much improved
17. Rheumatic heart disease	Much improved
18. Arteriosclerotic heart disease	Much improved

dure on alcoholism approximated the medical statistics.

There were 18 deaths in the group of 398 cases. Five (27%) were due to chronic alcoholism, with hepatic failure in three instances. The causes of death and the alcoholism recovery scale are listed in Table IV.

Another tangible result of the company program on alcoholism has been the reduction in absenteeism in the cases involved. The average recorded sick absence in the consultation clinic cases for a three-year period prior to referral was approximately 14 days per year. We have reason to believe that the absences due to alcoholic episodes themselves were masked as medical conditions. With treatment, the absence severity rate has been less than four days per year in the rehabilitated cases.

Summary

The long-term experience with the Consolidated Edison Company Procedure on Alcoholism in the 10 years from 1948 to 1957 indicates that 51% of the employees with a drinking problem have been able to maintain their jobs. More specifically, those who had the benefit of special medical treatment demonstrated that nearly 60% were successfully rehabilitated when reviewed over a follow-up period of more than five years. There has also been a reduction in absenteeism in the rehabilitated cases. These facts would indicate that a company program is a positive step toward the prevention of disability from chronic problem drinking.

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The Treatment of Snakebite

A. C. Stimson and H. T. Engelhardt, M.D., Houston, Tex.

DEATH from snakebite is uncommon. Swartzwelder[1] reports on a series of 306 cases of snakebite which he observed in Charity Hospital, New Orleans, from 1907 through 1946. It is of interest that in this study only two of the cases terminated fatally and that the last recorded death occurred in 1921. Although more snakebite fatalities are recorded in Texas than in any other state, only 18 deaths were recorded during the period 1950-1954. For this same period, 71 died from snakebite in the United States, while 144 died from stings and bites of venomous insects and animals other than snakes.[2]

The poisonous snakes in the United States are as follows: the water moccasin, or cottonmouth (Agkistrodon piscivorus); the copperhead (Agkistrodon contortrix); the rattlesnake (Crotalus); the pygmy, or ground, rattlesnake (Sistrurus); and the coral snake (Micrurus). The most dangerous of all these are rattle-

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Adult Water Moccasin

Copperhead

Western Ground Rattler

Canebrake Rattler

Coral Snake

Houston Zoo Photos, by Werler

snakes, which, with few exceptions, are more active in the evening and at night. During the cooler months of the spring and the fall, their habits are diurnal in nature, and one can expect to find them in the daytime as well as in the evening. Ordinarily in hot weather, they are quiescent, and during the heat of the day they seek cover from the sun in some shady place. Occasionally, they may be found in the summertime near golf courses where the grass has been watered and it is shady.

Most snakes, particularly the venomous ones, are chiefly active at night. Thus, there is increased danger of injury during the hours of darkness. It has been observed (Swartzwelder) that the period between April and October is the time of greatest exposure. This period coincides with warmer weather and with increased outdoor activity such as hunting, farming, gardening, and vacationing.

It is just as important for the physician to obtain a careful history when treating an individual who has been bitten by a snake as it is when treating any other abnormality or disease. We feel that it is most important that the physician determine that the patient has actually been poisoned, even though the patient brings a venomous snake with him.

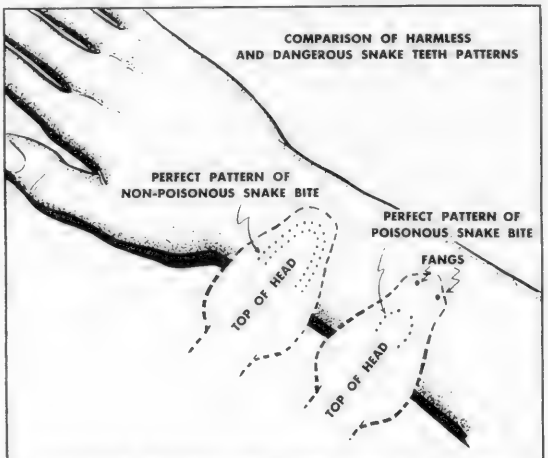
In instances where the offending creature is not available, the physician must attempt to determine if the symptomatology is due to a snakebite, or something else, such as the bite or sting of a spider, scorpion, or insect. Occasionally, such determination is impossible. Certain observations, however, help one make a specific diagnosis. It is known that snake envenomation usually results in a gradual increase in pain, while in other bites or stings there is usually a regression of pain with the passage of time. The exceptions are in bites of the black widow and its close relatives, the brown widow and the red-legged widow.

Occasionally, an individual is bitten and presents no clinical evidence of poisoning.[3] One might properly ask, then, why there is no poisoning, if there are visible punctures and the snake has been correctly identified as a venomous one. Several possibilities present themselves: First, all snakes shed their fangs at indefinite intervals and replace them with new fangs. It is possible that, during the interval, the snake may have been unarmed. This condition is unlikely, however, since the new fang drops into place almost immediately; indeed, sometimes before the old one is discarded. We have seen snakes with three fangs in place, occasionally four, and often only one (Fig. 1).

Second, illness of the snake may have a great deal to do with the potency of its venom.

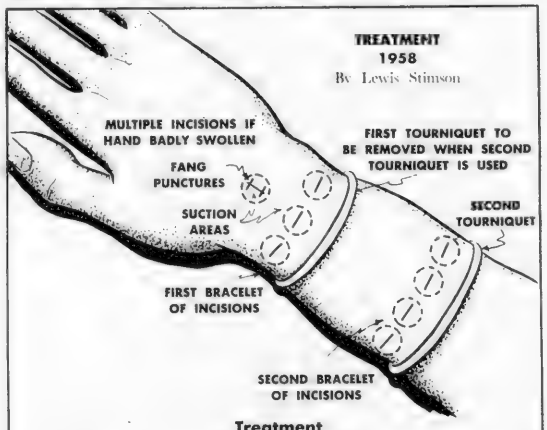
Third, the snake may have been so teased as to have largely dissipated its venom before biting. More frequently though, the snake loses most of its venom on the apparel of the victim, and merely touches the skin with the point of the fang, but not so deeply as to embed the poison-conducting orifice.[4]

The severity of the snakebite is influenced by the age and size of the victim. Bites in


COMPARISON OF HARMLESS AND DANGEROUS SNAKE TEETH PATTERNS
PERFECT PATTERN OF NON-POISONOUS SNAKE BITE
PERFECT PATTERN OF POISONOUS SNAKE BITE
FANGS
TOP OF HEAD
TOP OF HEAD

Comparison of Harmless and Dangerous Snake Teeth Patterns

This drawing illustrates perfect patterns. These occur in about 15-20 per cent of the total number of accidents. Usually, the wounds appear as numerous scratches or several punctures and often as only one indentation. They are very hard to detect on the body of a dark-skinned individual.



Treatment

The bracelets of incisions are linear instead of cruciate. The constrictors are applied loosely and moved three inches above the swelling as the venom advances proximally. Bracelets should follow swelling at intervals of about six or eight inches, in acute cases, until excessive swelling is retarded. Seldom, however, are more than one or two bracelets necessary if the wound is on an extremity and not the result of poisoning by a rattlesnake or large moccasin or unless there is undue sensitivity to the venom on the part of the victim. Suction should be continued for at least 8 hours and sometimes for as long as 24 hours.

Figure 1

the aged and very young usually carry a poor prognosis. Bites on the extremities are less dangerous, while bites on the face and neck are associated with greater danger. Shannon,[5] among others, implies sudden death may result from the injection of the venom directly into a vein. It is generally held that the larger the snake, the greater the amount of venom, although Stadelman[6] has recorded serious tissue destruction following the bite of even the youngest of pit vipers. It has been reported that a cobra has inflicted a fatal wound before fully emerging from the egg.

Generally speaking, snake venom is composed of complex proteins, which act as enzymes. Hyaluronidase is found in most venoms and is responsible for the quick spread of the venom along the lymphatics. Interestingly enough, the venom is rarely disseminated by the blood. The predominant venom of the pit vipers* is a hemotoxin which causes a hemolysis of the erythrocytes and a disturbance in the clotting mechanism so that, at the site of the bite, there is ecchymosis, edema, and tissue destruction.

The coral snake, a member of the Elapidae, secretes a venom in which a neurotoxin predominates. Death from such a snakebite is usually the result of bulbar paralysis.[7, 8]

We challenge the deadliness of the coral snake's bite in this country, as so often advanced. Of the several hundred cases of snake poisoning that the senior author has helped treat, he has seen nine caused by Micrurus fulvius Tenere bites. Not one of these was as serious as the least of the pit viper cases. Parrish[9] writes that he has treated five cases of coral snake bite, none of which was dangerous. Extensive correspondence with hospitals and physicians, within the territory where Micrurus ranges, has resulted in but one report of a death caused by a coral snake, and we feel that serum sickness or overexertion may have played an important role in this death.

A venomous snakebite wound first appears as a small white wheal surrounding each puncture. The wheal usually progresses to red, and later may turn blue, green, or purple. Depending on the nature of the venom, edema is present in varying degrees at the site of the infection, and the patient complains almost immediately of an intense burning pain at the site of the bite.

Shock may be present in various expressions, for example, a thready, weak pulse, cold, clammy perspiration, and faintness. Even syncope may result. Because the venom is spread by means of the lymphatic system, the regional lymph nodes usually become involved and enlarged; and, depending on the site of the bite, there may be involvement of the epitrochlear region, the axilla, the popliteal space, or the inguinal region. On occasion, the patient may present difficulty in breathing, which in some cases may actually be asthmatic in nature. In severe cases of snakebite, nausea and vomiting are common, and there is often conspicuous muscular twitching. It might be stated parenthetically that it is not uncommon to observe many of these symptoms in a nervous, tense individual who has been bitten by a nonvenomous snake. A diagnosis is easily made, however, in the presence of definite local signs at the site of the bite, shock, or evidence of involvement of the vascular system, such as local edema or varying degrees of conjunctivitis.

At best, snakebite treatment is heroic, and it would be interesting to learn of the number of persons who have undergone this treatment after having been bitten by nonvenomous snakes. The senior author has observed several hundred cases of snakebite in the past nine years. Among these, there have been many "false alarms"; and in many instances of actual bites by copperheads, moccasins, pygmy, or ground, rattlesnakes, and Texas coral snakes, no treatment was needed except an injection of tetanus antitoxin or toxoid and sedation because a detailed history and physical examination revealed no indications of poisoning.[10]

We are impressed with the damage caused by well-meaning individuals whose attempts at first aid result in brutally-deep incisions and tourniquets applied too tightly and for too long a period of time. These measures result in damage which in many instances exceeds that of the bite itself.

Contrary to public opinion, snakebite is not necessarily such a serious thing. Few deaths occur in spite of "woods-surgery" as practiced today or even with methods used 40 years ago, such as applying a poultice of half a freshly killed chicken, searing the wound with a hot iron, burning it with gun powder, or drinking a cocktail consisting of a quart of a mixture of lard, vinegar, quinine, boiled tobacco juice, and whiskey (Swartzwelder).

We believe that the sacrifice of surgical cleanliness exposes the patient to far greater danger than the time involved in transporting him to medical facilities, if a physician or hospital can be reached within an hour and if the wound is on an extremity.

Suction by mouth is dangerous, not, as so often suggested, to the operator but to the

*All of the poisonous snakes mentioned in this article except the coral snake are pit vipers.

patient. It is common knowledge that the human bite frequently results in a severe infection. This danger is magnified in the edematous, hemorrhagic wound following snakebite. One should not hesitate to use mouth suction, however, in the event of poisoning by the bite of a large (greater than 3½ feet in length) rattlesnake or water moccasin, or, any venomous snake, on the face, neck, or trunk. In such desperate cases, no time should be lost. Lacking other aids, a sharp piece of tin or broken glass may be sterilized by being passed through a flame and used as a cutting instrument, and a pliant vine used as a constrictor might save a life.

As a matter of principle, we are opposed to so-called "woods-surgery," unless, of course, the accident occurs in a remote area. By "woods-surgery," we mean the holding down of a victim by companions or good-intentioned neighbors and the use of one of the many snakebite kits now on the market, when medical aid is within an hour's drive. We believe that such treatment exposes the patient to far greater danger than if time were sacrificed to surgical cleanliness. The senior author has frequently observed children who have been subjected to such treatment only to find that the cause of the trouble was a nonvenomous snake.

What One Should Do When Bitten by a Snake

1. Kill the snake if it can be done within 10 minutes; and take its body to the physician.
2. If bitten on the extremities, apply a constrictor 3 inches above the wrist or ankle, as the case may be, so loosely as not to retard the venous return, and another constrictor above the elbow or knee. If the extremity turns white or blue, the constrictor is too tight. Release both constrictors at intervals of 20 minutes, leaving them off for 3 minutes.
3. Wrap cracked ice in a cloth and apply it to the wound. Do not allow the ice to come into direct contact with the flesh.
4. Drink strong coffee or a cola beverage if it does not nauseate (a teaspoon of spirits of ammonia is better if it is available).
5. If alone, walk slowly toward help; if with companions, have them carry you to a vehicle and proceed to a physician or hospital as fast as possible.
6. At all times, be as calm as the situation will permit.

What One Should Not Do

1. Do not get hysterical. Remember that few snakebites result in death.
2. Do not drink alcoholic beverages.
3. Do not attempt surgery or allow anyone other than a physician to do so, except as in the emergencies mentioned above.
4. Do not allow anyone except a physician to administer an antivenomous serum, except in dire emergencies, as mentioned.
5. Do not "squeeze" the wound to stimulate bleeding.

What the Physician Should Do When Confronted by a Case of Snakebite

1. Immediately upon the patient's arrival, remove all tight strictures and apply loose ones.
2. Sedate patient, if necessary.
3. As soon as possible, obtain, count, and type the blood. Order a urinalysis, and test the patient for sensitivity to antivenomous serum.
4. Measure the circumference of the limb at a point 3 inches above the wound. Record of the spread of edema should be kept hourly.
5. Under local anesthesia, connect the fang punctures with a ½ inch incision as deep as the considered depth of the wound. Thus the venom MAY be reached. Owing to the curvature of the fangs and the directional attack of the snake, the chances are great that the pocket will not be drained. Shannon writes that because of the "spreading factor," the fang punctures should not be incised. It is his belief that very little, if any, venom can be drained at the site of the bite. We agree, however, with the late Dudley Jackson,[11] who felt that the fang punctures should be drained. We advise then: drain fang punctures as soon as possible and not later than four hours after the accident.
6. In proportion to the seriousness of the poisoning, make supportive incisions in areas of excessive edema, not only proximally, but also distally. Such measures will minimize the danger of sloughing. We have found that linear incisions stimulate drainage much better than X-type incisions, and good drainage is paramount.[11] We agree with Jackson,[11] who wrote that "one incision will heal as quickly as several, and further, *under*-treatment is far more dangerous than *over*-treatment regarding incision and suction." The incisions should be 1 inch apart and ⅛ to ¼ inch deep. The length of the incision depends upon the size of the suction cup, and the depth should be great enough to secure adequate drainage of the lymphatics (Figs. 2, 3, and 4).
7. Apply suction cups steadily for the first two hours; then remove them for 15 minutes each hour. Lay warm saline dressings lightly

Fig. 2—Typical poison snake wound. Picture taken 14 hours after accident. Practically no treatment prior to that time. Lancing and suction applied after 14 hours. Patient released from hospital in 48 hours and made an uneventful recovery.

over each incision during the rest period. Continue suction for at least eight hours in moderate cases and many hours in critical ones. As long as the swelling advances to the extent of ¼ inch per hour, continue suction with bracelets of incisions at 3-inch intervals proximally (Fig. 1). In critical cases, or even in serious ones, bracelets of incisions are indicated. Cuts should be made at intervals of 2 inches proximally and circumscribing the limb equidistantly. Care should be taken that the incisions in the bracelet are not closer than 2 inches.

8. After the edema has receded to the physician's satisfaction, wrap the limb in a towel containing cracked ice for an indefinite period, depending upon the patient's systemic condition. We agree with Shannon in regard to the danger of cryotherapy as advocated by Sthanke.[12]

9. Test the patient for individual sensitivity to antivenomous serum. Even though the skin and eye tests are negative, there is always the chance of serum sickness. Antivenomous sera should not be prescribed except in quite severe poisonings. The reactions are sometimes much more serious than the venom itself. If envenomation is so serious as to demand its use, it should be administered in relatively vast quantities—60 cc to 120 cc. While we are quick to admit that it certainly has an important place in medical science, we are fearful of its side effects if used promiscuously. Antihistaminics in adequate doses should always be given concomitantly.

Corticotropin (ACTH), cortisone, antihistaminics, and streptomycin and other antibiotics

Fig. 3—Demonstrating the use of suction cups.

Fig. 4—Same patient as in Fig. 3 after removal of suction cups.

have been used with questionable results.[13] Trypsin and curare are now in the experimental stage. We have found calcium gluconate administered intravenously to be of great help in severe cases. Tetanus and gas gangrene antitoxin as well as antibiotics should be administered.

Summary

The rarity of fatal snakebite in the United States is pointed out. First aid and definitive treatment is outlined. It is pointed out that antivenomous serum may be more dangerous than the venom itself.

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SMOKING AND LUNG CANCER: RECENT EVIDENCE AND A DISCUSSION OF SOME QUESTIONS. Jerome Cornfield et al. 1959. 31 pp.
Reviews some of the more recent epidemiological and experimental findings on the relationship of tobacco smoking to lung cancer, and discusses some criticisms directed against the conclusion that smoking has a causal role in the increase of bronchogenic carcinoma.

UTERINE-CANCER MORBIDITY IN NEW YORK CITY AND ITS RELATION TO THE PATTERN OF REGIONAL VARIATION WITHIN THE UNITED STATES. William Haenszel and Margaret Hillhouse. 1959. 25 pp.
The results of a New York City uterine-cancer morbidity survey are presented. The results were generally consistent with observations reported from other northern areas of the United States. The collective findings of morbidity surveys and related studies within the United States point to a regional difference in cervical cancer but not in corpus cancer, the cervical rates being higher for white women in the south.

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This booklet was prepared to help the physician show what can be done for the stroke patient at home. Pictures and text provide information suitable for health education purposes.

LITTLE STROKES—HOPE THROUGH RESEARCH. (PHS Publication No. 689). 1959. 15 pp.
Describes how prompt diagnosis and early treatment of "little strokes" often avoid more serious paralyzing attacks.

SELECTED REFERENCES ON CARDIOVASCULAR DISEASE: AN ANNOTATED BIBLIOGRAPHY FOR NURSES. (PHS Publication No. 472). 1958. 72 pp.
References are arranged in sections according to the main types of cardiovascular disease and other major interests in the field, such as the public health and rehabilitation aspects. Final section lists films and other audiovisual aids.

The Effect of Cold on Manual Performance

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THERE are many important practical aspects to the effect of cold on manual performance, although the present extent of knowledge in this field is surprisingly meager.

For example, as long ago as 1922, Osborne and Vernon showed the importance of the influence of temperature on accident frequency when they published their observations of industrial accidents made during the first World War. They found that minor accidents (cuts) incurred by munition workers engaged on light or moderately heavy work were lowest when the environmental (dry-bulb) temperature was between 18.5°C (65.3°F) and 20.5°C (68.9°F), while they were 23% higher at temperatures greater than 25°C (77.0°F) and 34% higher at temperatures lower than 13°C (55.4°F). Vernon (1940) has suggested that the higher accident rate at low temperatures was probably due to a reduction of finger dexterity, as the workers studied were frequently handling cold metal objects.

Further examples of more recent origin help to illustrate other aspects of the problem. It has been reported by McCance, Ungley, Crosfill, and Widdowson (1956) that during World War II, of 2,800 men in ships which sank in sea temperatures below 10°C (50°F), 30% died before reaching a boat or raft. This percentage would certainly have been higher but for the fact that many of the ships in northern waters were traveling in convoys and rescue was rapid. Most of the men died while in the water rather than in the parent ship, but it is not possible to say how far death was due to inability to swim, to systemic effects of hypothermia, or to local effects of cold on the limbs.

The extent of reported cases of frostbite during the Korean campaign (Orr and Fainer, 1952; Schuman, 1954) also underlines the need for further understanding of the various factors contributing to incapacitating cold injury and the effectiveness of different countermeasures, including protective clothing. Hands provide a special problem in this respect as they "cannot be kept warm in arctic environments except by maintaining a rapid circulation of blood (by exercise), by withdrawing the hand and arm into a parka type shirt, or by artificial heat" (van Dilla, Day, and Siple, 1949).

Accident surveys and injury reports, however, can only indicate the need for serious investigation of the problem and suggest the variables likely to be involved. Experimental analysis is also required to enable the optimum range of environmental conditions for unimpaired manual performance to be defined and to assess the influence of subtler factors, such as individual differences in susceptibility to the deleterious effects of cold exposure.

To date, experimental study of the effect of low environmental temperatures on manual performance has been confined almost entirely to the investigation of sensorimotor tasks and muscle strength. There are obvious difficulties in studying the effect of cold on certain other aspects of voluntary movements, as, indeed, there are in investigating the factors involved in the initiation and control of voluntary movements in man under normal climatic conditions. Most of these studies have been published during the last 10 or 15 years, but so far as the present authors are aware, only certain aspects of these reports have previously been reviewed (Russell, 1957: Provins, 1958).

Reaction Time

The first study relating variations in reaction time to changes in body temperature was published by Kleitman, Titelbaum, and Feiveson (1938). These authors investigated the diurnal variation in performance of both simple and choice reaction times to visual and auditory stimuli in five subjects. The oral temperature was recorded by clinical thermometer after each test. The results suggest that there may be an association between body temperature

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and reaction time, although the variations recorded in both were extremely small. The lowest reaction times were recorded when the average oral temperature was highest, and the effect was apparently more marked in the choice reaction time situation. A further study by Kleitman and Jackson (1950) on the diurnal variation of performance in nine subjects confirmed this inverse relation between body temperature and choice reaction time performance.

However, other studies designed to investigate the effects of short or prolonged exposure to low environmental temperatures on human performance have failed to detect any significant increase in simple or choice reaction time. Unfortunately, not all the investigators have recorded body temperature, and in all studies reviewed the subject has been protected from the cold by wearing heavy clothing.

For example, Williams and Kitching (1942) tested both simple and choice reaction time to visual stimuli (and in some instances, sound) at climatic chamber temperatures of –18°C (–0.4F) and –45.5°C (–49.9°F) during at least one hour's exposure. Only a very few subjects were employed, and, although both skin and rectal temperatures were recorded throughout, no comparison was presented between the measures of body temperature and reaction time for each subject. However, the authors conclude that there was no direct association between reaction time and body temperature and that the variations in reaction time which did occur could be better explained in terms of distraction due to the discomfort experienced.

Horvath and Freedman (1947) investigated the effect of exposure to a temperature of –29°C (–20.2°F) on visual choice reaction time in 22 subjects. The men lived in these conditions continuously for a period of 8-14 days and were given a series of 50 test responses three times a day. No significant change in the average reaction time for each subject was recorded due to the exposure, although tests of dexterity and hand strength were affected.

The effect of cold on the reaction time involved in pressing finger triggers of different loading or resistance has been investigated by Craik and Macpherson (1943) and is discussed in the section on muscle strength.

The most recent investigation of the effect of cold on reaction time is the study by Teichner (1958) who exposed groups of soldiers to a variety of combinations of air temperature and wind speed, the most severe being –34.5°C (–30.1°F) and 20 mph. Subjects were given tests of simple reaction time after 45 minutes exposure and again following a short mild exercise after 65 minutes exposure to the same conditions. The results indicated that at low wind speeds (<5 mph) low environmental temperatures have no effect on simple reaction time at least down to –34.5°C (–30.1°F) but that at wind speeds of 10 mph or more, low ambient temperatures produce a significant increase in the time taken to respond to a simple stimulus. However, although skin temperatures were recorded from a number of body areas, no correlation was found between body temperature (based on a mean area-weighted skin temperature) and reaction time.

The results of all these reaction time studies suggest that at low ambient temperatures there may be some impairment of performance, particularly if there is local cooling of the hand and the response mechanism requires more than a light operating pressure. There is likely to be an increase in reaction time with a lowered body temperature, although some impairment may be recorded without, probably due to an increased awareness of discomfort or a decreased awareness of other, more general, environmental stimuli. On the other hand, it has been suggested that at only slightly reduced ambient temperatures there may be an increase in general alertness (Barmack, 1939). Clearly, more experimental evidence in this field is urgently required.

Tracking Proficiency

There are few studies of tracking proficiency in the cold, but in contrast to the results of reaction time experiments, nearly every investigation has provided evidence for appreciable impairment of performance at low ambient temperatures.

In the study by Teichner and Wehrkamp (1954), subjects (clothed in U.S. Army fatigue uniform) were tested on a Koerth-type pursuit rotor after 20-30 minutes' exposure to an ambient temperature of 13°, 21°, 29.5° or 38°C (55.4°, 69.8°, 85.1°, or 100.4°F). The results not only indicated an impairment of performance above and below 21°C but also showed a sharper decrement at the lower temperature. In a further study, by Teichner and Kobrick (1955), five subjects (clad only in shorts, T-shirts, and socks) lived in a constant temperature chamber at 13°C (55.4°F) for 12 days and were tested daily on the same tracking task as before. An impairment of performance was again recorded at the lower temperature, although some recovery was made during the 12 days' exposure. However, evidence is presented which suggests that the impairment was the

result of a lowering of the final limit of performance rather than a reduction of the rate of acquisition of the skill.

Russell (1957) investigated the effect of exposing subjects (wearing arctic protective clothing but with bare hands) to different environmental temperatures down to –10°C (14°F). Their performance was tested on a variety of tasks at 20-minute intervals during a total exposure of just over an hour. Two measures of tracking proficiency were employed, both of which showed an increasing impairment of performance at ambient temperatures below about 10°C (50°F) although no systematic effect of the duration of exposure was found. A further study was undertaken by Newton, Meketon, Roote, and Stargel (1958) specifically designed to investigate any differential effect of cold on the type of control lever employed in testing tracking proficiency. These authors exposed 12 subjects (who wore parkas) for approximately 30 minutes at different ambient temperatures down to –20°C (–4°F) and in each temperature condition tested their performance on a compensatory tracking task using either a "pressure" or a "free-moving" control operated by pronating and supinating the hand. Although no over-all effect of cold on tracking performance was found on either task, a differential effect of type of control and temperature condition was evident which was dependent on the subject employed. It seems likely that the lack of an over-all temperature effect was due to the short duration of the exposure and the heavy clothing worn.

A recent investigation by Payne (1959) on the effect of low ambient temperatures and administration of glycine on tracking proficiency using the U.S.A.F. School of Aviation Medicine Multidimensional Pursuit Test (Cm 813 E) showed a significant variation in performance with temperature condition but not with the glycine treatment employed. In this study, the subjects (dressed in fatigue uniforms) were exposed for approximately 3 hours 20 minutes to an ambient temperature of 4.5°, 13° or 21°C (40.1°, 55.4°, or 69.8°F). Appreciable impairment of performance was recorded for the lowest temperature condition investigated (4.5°C—40.1°F) and a somewhat paradoxical peak of proficiency at 13°C (55.4°F), although it seems likely that this 13°C (55.4°F) peak may be due (in some measure) to the method of treatment of the results.

Tracking tasks therefore (which can be considered to provide a serial reaction time situation) may be expected to suffer some performance decrement at temperatures below about 10°C (50°F) even when the subject is wearing specially designed protective clothing (except for bare hands). A similar decrement may be recorded in a shorter period of time without any special clothing or a more marked impairment with severer exposure conditions.

General Dexterity

There are a large number of investigations concerned with the effect of cold on performance of specific manipulative tasks which are difficult either to classify or to interpret in terms of fundamental processes. Frequently, such studies have a distinctly practical orientation toward a particular military or industrial situation, and for this reason they are usually of relatively little general interest. Only a few of the more important and accessible publications have therefore been selected for review here.

In the practical situation, personnel likely to be subjected to prolonged cold exposure are equipped with special protective clothing. The wearing of gloves however, is likely to cause a distinct impairment of manual dexterity (Rubin, 1957), and for this reason it may be desirable for particular tasks to be carried out with the bare hands. It is then essential to know to what extent local cooling of the hands affects manipulative ability and to what extent it is dependent on the general body temperature.

Springbett (1951), using the Minnesota Manual Dexterity Test and two other measures of motor performance, was the first to investigate the effect of local and general body cooling on manual dexterity. Sixteen male subjects (wearing normal indoor clothing) were exposed to ambient conditions of about –4°C or 21°C (24.8° or 69.8°F) (with separate temperature control of the hand and forearm) for up to 70 minutes and tested on each task both before and after exposure. It was found that "subjects with cold hands showed significant impairment, whether their bodies were warm or cold and conversely, subjects with warm hands showed no impairment, whether or not their bodies were warm or cold." These results have been confirmed by Gaydos (1958) and Gaydos and Dusek (1958), using two other tests of manual dexterity (knot tying and block stringing). These authors found a significant impairment of test performance when the hand skin temperature was lowered to about 11.5°C (52.7°F) but no decrement when hand skin temperatures were maintained at 27°C (80.6°F) or higher, despite a general cooling of the body surface to 25.5°C (77.9°F) in both

cases. Conversely, performance on the two tests appeared to be equally bad whether only the subject's hands were exposed to cold or whether he worked *in toto* in a low ambient temperature.

In an attempt to determine more closely the cause of the impairment of dexterity with local exposure to cold, Hunter, Kerr, and Whillans (1952) investigated the speed of flexion of the index finger at the proximal interphalangeal joint. Three methods of exposure to cold were employed: (1) local chilling of the joint, (2) chilling of the hand up to the wrist, and (3) chilling of the hand and arm up to the elbow. Chilling was achieved by immersion of the part concerned in cold water until the skin temperature over the proximal interphalangeal joint was 12°C (53.6°F). The result showed a significant reduction in the number of flexion movements (involving maximum flexion of the joint) completed in a 10-second test period after chilling by each method. The differences, however, between the means recorded for each procedure were not statistically significant, and the authors concluded that the impairment in performance must therefore originate in the region of the joint itself. Further study of the characteristics of joint movement led the authors to propose that the impairment of joint mobility in the cold may be due to an increase in viscosity of the synovial fluid.

In following up this work, Le Blanc (1956) compared the effect of immersing the finger or hand with immersing the forearm only in ice cold water for 10 minutes. Besides testing the subjects before and after exposure on the test of finger flexion used by Hunter et al., a simple tapping task was also employed which involved only 15° flexion of the joint. Cooling the arm but leaving the hand exposed to a comfortable air temperature (about 19.5°C —67.1°F) produced a considerable impairment of performance on both tasks, while exposure of the finger or hand alone caused about the same degree of impairment on the test involving maximum finger flexion but a much smaller decrement in tapping performance. The results therefore suggest that, while tissues in the region of the joint may limit the extent of finger movement in the cold, cooling the structures of the forearm may cause an equivalent or even greater decrement in dexterity, presumably due to interfering in some way with activity of the appropriate muscles.

In a study recently published by Clarke and Hellon (1959) the degree of hyperemia produced by rhythmic isometric contraction of the forearm muscles at a rate of 60 per minute was measured with the arm and hand separately immersed in water at temperatures of 18° and 34°C (64.4° and 93.2°F). It was found that changing the temperature of the hand had no pronounced effect but that cooling the forearm appreciably increased the hyperemic response. On the assumption that post-exercise hyperemia is related to the total energy conversion of the muscles, the authors suggest that the results are explicable on the basis of an increase in viscosity in the forearm muscles on cooling. As the exercise involved little or no actual movement of the fingers, the absence of an increased hyperemic response on cooling the hand alone confirms and extends Le Blanc's conclusion that impairment of joint function is related to the degree of finger movement.

The relationship between impairment of joint flexibility and impairment of general finger dexterity has been investigated by Coffey (1955 *a*). He found that manual dexterity (measured by the Craik screw-plate) declined progressively with joint surface temperature below about 6°C (42.8° F), together with a similar but more marked decrease in joint flexibility. However, Teichner (1957), in an investigation of the effect of different combinations of low air temperature and wind speed on another test of dexterity (Minnesota Rate of Manipulation Test), found no significant correlation between impairment of performance and either digital cooling rate or finger skin temperature. On the other hand, this author points out that, owing to the small size of his sample, the lack of significant relationships cannot be accepted with confidence.

These results therefore suggest that local cooling of the hand or arm usually produces a significant impairment of manual dexterity irrespective of the general body temperature (within limits). Cooling of the hand or finger alone produces joint stiffness and restriction of joint movement but local cooling of the forearm may cause a more severe impairment of dexterity which is probably due to an increase in muscle viscosity of the long flexors and extensors of the fingers. Conversely, a reduction in general body temperature may not be accompanied by a reduction in manual dexterity so long as the hands and arms are kept warm.

Tactile Discrimination

A wide variety of tests of tactile discrimination and kinesthetic sensitivity have been devised to assess impairment of function due to cold exposure (Weitz, 1941; Bartlett and Gronow, 1952; Mackworth, 1953; Teichner and Zigler, 1953; Russell, 1957).

The effect of a lowered skin temperature on the threshold to a vibrating stimulus was studied by Weitz (1941), using a small area on the forearms of six subjects. The results showed an increase in threshold to a stimulus of 256 c/s with reduction of the skin temperature by from 4.5°C to 13°C (40.1° to 55.4°F) although the degree of sensory impairment for any given temperature change varied from one subject to another. This deterioration in vibration threshold during exposure to cold is confirmed by the work of Mackworth (1955, 1956). He found an appreciable increase in threshold value to a 150 c/s stimulus during only six minutes' exposure of the index finger to still air at −16° to −25°C (3.2°F to −13°F), and a much more marked deterioration in sensitivity with a slight air movement of 4 mph. These results appear to be accompanied by a simultaneous fall in finger skin temperature.

As another means of assessing tactile sensitivity and one which could be readily employed in field work, Mackworth (1953) devised the V test. This is essentially a test of two-edge discrimination and correlates well with other sensory tests especially two-point discrimination (Bartlett and Gronow, 1952). In his initial series of experiments, Mackworth (1953) showed that severe impairment of two-edge discrimination could be produced by only three minutes' exposure to cold conditions and that the effects of raising the wind speed from still air to only a few miles an hour were just as important as the effects of lowering the air temperature by 5°C (9°F). Moderate winds also greatly intensified the numbing effects of the lower air temperatures employed −30° to −35°C (−22°F to −31°F). Plotting the average V test results for all subjects against finger skin temperature, Mackworth found a close association between the two, which appeared to be almost linear below a skin temperature of 20° to 25°C (68° to 77°F). In a later experiment designed to study this association more closely, Mills (1956) found that the log of the average two-edge threshold for all subjects was linearly related to finger skin temperature. In other words, as the skin temperature decreased, the loss of tactile discrimination was small at first but increased rapidly as freezing point was approached. However, it has since been shown by Morton and Provins (1959) that if differences between subjects in the acute exposure situation are taken into account, the association between finger numbness and skin temperature is quite different. These authors found that the deterioration in tactile discrimination is much more critically related to skin temperature and that the typical association is expressed by an L-shaped curve. These results were confirmed by Provins and Morton (1959) under more closely controlled experimental conditions using water instead of air as the medium of cold exposure. Five subjects immersed an index finger at each of six water-bath temperatures ranging from 2° to 30°C (35.6° to 86°F) for 20 minutes, during which finger skin temperatures were recorded continuously. After 15 to 20 minutes' immersion (when the finger was in thermal equilibrium with the water), there was little impairment of two-edge discrimination at temperatures of 6°C (42.8°F) or higher. However, at 4°C (39.2°F) there was marked impairment and at 2°C (35.6°F) all subjects experienced complete numbness at the test site. In these experiments, spontaneous rewarming of the finger due to cold vasodilatation of the digital vessels was prevented by inflating an arterial occlusion cuff. In another series of experiments reported in the same paper, these authors showed that under normal circumstances, immersing the finger in ice-cold water for 40 minutes produced a cyclic rise and fall in tactile discrimination corresponding to the appropriate phases of the "hunting reaction," first reported by Lewis (1930).

Thus, there is a definite impairment of tactile discrimination with exposure to cold that can be related to the temperature of the part concerned, i.e. the fingers. If the hand is being cooled, finger sensitivity is likely to be better than the skin temperature of the finger might suggest, while if the hand is rewarming, sensitivity may be worse than predicted. Nevertheless, with a finger temperature of 6°C (42.8°F) or higher there is likely to be very little impairment of tactile discrimination, although below this temperature level a very marked reduction in sensitivity may be expected.

Muscle Strength

The first investigation of the effect of cold on muscle strength was reported by Craik and Macpherson (1943) on two subjects. They found that immersing the hand for 15 minutes in water at 7°C (44.6°F) reduced the mean hand grip strength by 21% and the strength of opposing the fingers to thumb by 44%. The former movement is achieved mainly by forearm muscles which were clothed and presumably cooled less than the small muscles of the hand employed in opposition of finger and thumb. This suggests that strength, like dexterity, is mainly affected by local cooling of

the limb rather than by general body cooling.

However, Horvath and Freedman (1947) in a study of 70 soldiers who were dressed in heavy protective clothing (including two layers of mittens), found an average decrease of about 28% in hand grip strength after the subjects had been sitting quietly for three hours in a cold room at an ambient temperature of about –25° (–13°F). Russell (1957) also found a decrease in hand grip strength in subjects (clothed in parkas) who were exposed to ambient temperatures of 0°C (32°F) or lower for about an hour, but in this experiment the hands were left bare.

In recent studies by Wright (1959 *a*, *b*) of the factors influencing diurnal variation in the strength of hand grip, this author found an appreciable change in grip strength which corresponded closely with the diurnal variation in body temperature. Four different types of grip dynamometer were used and starting tests at different times or staying awake all night did not alter the diurnal pattern. In contrast, the effect of inducing a rise in body temperature by hot baths did affect grip strength.

That muscle temperature is probably the critical factor has been shown by Clarke, Hellon, and Lind (1958). These authors found that immersion of the forearm in water at 18°C (64.4°F) or more (up to 42°C—107.6°F) for 30 minutes made little or no difference to the maximum tension which could be exerted on a strain-gauge hand-grip dynamometer by an attempted flexion of the fingers. However, at water-bath temperatures below this value (corresponding to a muscle temperature of about 27°C (80.6°F) a progressive impairment of grip strength was recorded until at 2°C (35.6°F) it had declined to 40% of the control value.

The effect of cold on the ability to maintain a submaximal contraction has also been investigated by Clarke et al. (1958). In this series of experiments, the duration of submaximal contractions held to the point of fatigue was measured on the hand-grip dynamometer for 4 subjects after they had immersed their forearms for 30 minutes in water at temperatures between 2° and 42°C (35.6° and 107.6°F). Contractions were longest after the arm had been immersed in water at 18°C (64.4°F), above and below which temperature the duration achieved was progressively shorter. In a further series of experiments, Lind (1959) compared the effect of immersing the forearm in water for 30 minutes at either 18° or 34°C (64.4° or 93.2°F) on the ability to maintain a submaximal contraction to fatigue on 5 successive occasions when the interval between contractions was 3, 7, 20, or 40 minutes. The hand-grip dynamometer was again used and the results showed that the duration of contractions for a given interval between contractions was always greater in water at 18°C (64.4°F), although the duration of the final contractions fell off markedly at both temperatures when the interval between contractions was reduced from 40 to 3 minutes.

Limb strength is therefore likely to be impaired during cold exposure to an extent depending on the amount of local cooling of the muscles below about 27°C (80.6°F). Furthermore, the length of time for which a submaximal contraction may be maintained will also be reduced below this muscle temperature.

General Discussion

There are obvious dangers and difficulties in attempting to generalize from the evidence available in these relatively few experimental studies. For example, it was shown long ago by Perrin (1921) and Muscio (1922) that there is very little correlation between performance on one test of motor ability and another. Although this result is likely to depend on the particular tests employed and the degree of control exercised over the considerable number of experimental variables, it clearly suggests caution in the interpretation of performance on any one sensorimotor task in the cold. This point has recently received confirmation from the studies of Teichner, Kobrick, and Dusek (1954) and Rubin (1957).

Furthermore, the extent of individual differences in susceptibility to impairment of manual performance should not be underestimated. Apart from the variation between subjects referred to in studies mentioned earlier in the present review, differences in manipulative ability at low environmental temperatures have also been reported between groups of subjects classified on a racial or occupational basis. For example, Coffey (1955 *b*) administered a number of sensorimotor tests ("V" test, 'Craik screw-plate,' joint flexibility and strength of grip) to a group of 10 Eskimo and Indian young men in the North West Territories of Canada. In comparing their performance with that of a group of white males who had been in the north for about six weeks, Coffey states that "These measures all indicated that the native groups were definitely superior to the white groups: at comparable temperatures, the decline of each of these functions was, for the natives, about half as much as that reported by the white personnel." To what extent this

difference in ability at low temperatures may be due to the exercise of some special "cold-handling" skill is not clear, although the author attributes the difference to some more physiological cause. Confirmatory evidence for this view comes from the results of Mackworth's (1955) test of tactile discrimination at low ambient temperatures. In his initial series of experiments, he showed that air temperatures of −16° to −25°C (3.2°F to −13°F) at wind speeds of about 4 mph had a much greater numbing effect on the exposed fingers of men classified as "indoor" types than on the fingers of men who spent most of their working day out of doors in the Canadian sub-Arctic. Mackworth suggests that this difference can be attributed to a physiological acclimatization which may take the form of an increase in the resting finger blood flow, and provides some supporting evidence for this view. Certainly it has been shown that the resting hand blood flows and skin temperatures of Eskimos are considerably higher than those of Canadian medical students (Brown and Page, 1952) while other relevant aspects of local vascular responses to a cold stimulus in terms of individual and racial differences have been investigated by Bader and Mead (1950), Yoshimura and Iida (1950) and Meehan (1955).

Information of this sort is clearly important in the selection and training of personnel for manual work in low ambient temperatures. At the moment however, very little evidence is available on such points as, for example, whether persons who are highly dexterous on a particular task at normal air temperatures (about 20°C—68°F) are also above average on the same task in the cold, although the recent study by Morton and Provins (1959) provides a step in this direction. Furthermore, relatively little advance has been made in the understanding of individual differences in the ability to handle cold metal (without wearing gloves) since the original experimental studies of Lewis and Love (1926). Coffey (1955 *b*) has recently cited the case of a Loucheaux Indian who worked for about 6 minutes with metal tools at an ambient temperature of −40°C (−40°F) and a wind speed of 35 to 40 mph using his bare hands without suffering from any form of cold injury. Although this appears to be an exceptional feat (*cf.* Goldsmith, 1959), more detailed observations are necessary to determine if such a performance is due to acclimatization of the hands, local habits, racial factors, or some special cold-handling skill. It is outside the scope of this review to consider the etiology of cold injury from whatever cause, but clearly further evidence is necessary to clarify the conditions limiting safe handling of cold metal as it affects differences in manual dexterity.

No less important, of course, is the need to design equipment for use in the cold so that all handles and operating gear are large enough and conveniently shaped to be operated while wearing thick gloves. Where this is not possible, knobs and switches should be made with material of low thermal conductivity so that they can be safely operated with bare hands. In other words, every effort should be made to 'fit the job to the worker' as well as to select and train the man for the job.

[AUTHORS' NOTE: Since the above review was written, the proceedings of a conference sponsored by the Headquarters Quartermaster Research and Development Command (U.S. Army Quartermaster Corps, Natick, Mass.) held on April 23-24, 1956, have become available to the authors. Many important papers are included in the report, which is entitled "Protection and Functioning of the Hands in Cold Climates," edited by F. R. Fisher. Published by the National Academy of Sciences, National Research Council, Washington, D.C., 1957.]

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Nutritional Factors in Cold Acclimatization

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WHILE it has been difficult to establish definite and specific evidence of general physiologic acclimatization to cold in man, acquired through repeated or protracted cold exposure, there is clear-cut evidence of acclimatization in certain laboratory animals.

In the field of human physiology, our interest has been focused on the following questions: *(a)* What is the nature of the physiologic response to cold stimuli? *(b)* Does cold exposure modify the subsequent physiologic response? *(c)* How and to what extent is this modification beneficial? *(d)* If beneficial, what does it take in terms of conditioning, training, or accustomization to bring about this desired alteration? It is clear that if inherent or acquired differences in response to cold exposure do exist, an understanding of these differences would influence the selection and utilization of personnel for cold weather operations.

Individual differences in tolerance to cold exposure have been recognized ever since the time of the ancient Greeks.[1] It is difficult to evaluate many of the early reports of racial differences in endurance to cold exposure which concerned primitive groups of people—such as the Fuegians described by Darwin[2] or the Australian aborigines described by Hicks[3]—who were said to have lived naked or almost naked under fairly severe climatic conditions. The same is true of descriptions given by early travelers among certain Arctic or sub-Arctic tribes.[4]

Although Stefansson's statement[5] that the typical Eskimo lives under tropical or subtropical conditions has been widely quoted, the Eskimo is generally considered to be subject to considerable cold exposure. It would, therefore, be reasonable to assume that if natural human adaptation to cold does exist in one form or another, one would expect to find evidence of such adaptation in the Eskimos—who have successfully survived along the shores of the Polar Sea for several thousand years with at least their faces frequently exposed to considerable cold.

For the purpose of this discussion, it might be of interest to compare our findings in the Eskimos as well as in various groups of whites exposed for long periods to cold climates, with animals used in experiments on cold acclimatization. Since, in wild animals, natural adaptation to cold is accomplished through the increased insulation furnished by thickening of their pelts rather than through increase in metabolism,[6] a similar comparison between man and Arctic animals would be of less value.

Although this discussion will deal mainly with nutritional factors in cold acclimatization, reference will necessarily be made to such factors as the degree and duration of the environmental exposure; behavior, activity, and energy expenditure; energy metabolism; and thyroid activity—since all these factors have a bearing on nutritional requirements.

In most animal experiments, rats have been exposed to temperatures of around 5°C (41°F). Under these conditions, the rat—normally in thermal equilibrium at 21° to 25°C (69.8° to 77°F)—probably loses heat[7] at a rate of roughly 100 Cal/m²/hr and must more than double its metabolic rate in order to survive.

In rats kept at this temperature for more than four to six weeks, the following changes occur:[6, 8, 9] There is a marked increase in metabolism up to several times that of basal level, about 60% to 100% increase in food intake, increased cellular metabolism, increased vascularization of muscle tissue, increased thyroid activity, adrenal enlargement, increased ascorbic acid requirements, blood changes, and changes in water metabolism. The outstanding feature in the acclimatized rat appears to be its ability to maintain a very high metabolic rate; this ability is absent in the unacclimatized rat and consequently it is unable to survive.

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When comparing these facts from animal experimentation with findings in man habitually exposed to cold environments, the following differences are observed:

1. *Environmental Exposure*—The continuous exposure of rats to 5°C (41°F) is probably far severer than the environmental challenge to which even the most primitive native groups are exposed. This would be comparable in man to exposure of nude subjects to a temperature of roughly 5° to 10°C (41° to 50°F) continuously for several weeks.

In the case of the Australian aborigines, the air temperature only occasionally approaches this low level, and they then surround themselves with small campfires.[3] The clothing of the Eskimo, consisting of caribou fur in thicknesses of 1½ to 3 inches, has probably the equivalent of 7 to 12 clo, which would be adequate for maintaining thermal balance under practically all conditions encountered by him during outdoor exposure. On the basis of Belding's prediction[10] of total insulation required for prolonged comfort at various activities in the shade as a function of environmental temperature, it is estimated that an Eskimo would require up to 11 to 12 clo while sleeping and less than 4 clo while active, at −40°C (−40°F). This clothing requirement would be amply met by one to two layers of caribou fur.[11, 6] Similarly, the present Arctic uniform or flying clothing, providing somewhat less than 4 clo, would offer adequate protection during moderate or light work at −40°C when there is protection from the wind. Environmental conditions severer than this would be expected for only about a total of two days during the month of January in central Alaska.

Exposure of the unprotected face to convective and radiation heat loss would probably amount to less than 7 calories per hour, or less than 10% of the metabolism, if it is assumed that the exposed area is 300 cm^2 and the temperature of the skin 30°C (86°F). The maximum long-wave radiation under these conditions would be roughly 0.408 $Cal/cm^2/min$.

The added heat loss through the exchange of respiratory air in man exposed to cold is not of great importance. The respiratory heat loss is only 3 Cal/hr greater in a man standing at air temperatures of −40°C (−40°F) than at 0°C (32°F).[12] This difference is probably balanced by the difference in evaporative heat loss from the rest of the body.

A survey of ambient temperatures in different Eskimo dwellings during various seasons of the year in Alaska revealed that the mean indoor temperature is roughly 70°F during the day and about 50°F during the night, at which time they are covered by fur or regular bed clothing.[13] Apart from very brief periods of outdoor exposure (less than an hour or so per day) during bad weather, they spend on an average one to four hours daily outdoors during the winter and about five to nine hours in the summer.[14] Apart from brief exposure of the hands or fingers as needed for the performance of various tasks, only the face is constantly exposed for prolonged periods. The Eskimos' ability to withstand such exposure of the face is a phenomenon which should be studied in detail.

From the above summary, it appears that it is hardly justifiable to compare the Eskimo in his normal environment with the rat exposed to 5°C (41°F) when discussing general acclimatization to cold. Either the Eskimo would have to be exposed to severer cold or the rat to less severe cold—similar to that of the normal Eskimo environment—if one is to make valid comparisons.

2. *Behavior and Activity*—In the case of the Eskimo, it may be said that his success in combating the Arctic environment lies in his ability to avoid the extreme cold. It is characteristic for the Eskimo to maintain a steady, even, and rather moderate pace or level of activity, just sufficient to keep himself warm without exhausting himself. This level he can maintain almost indefinitely because of his stamina, achieved through training from childhood.

It should be emphasized that when men are exposed to cold under field conditions, it is usually a matter of combined exposure to cold and exercise, with a high level of physical activity. In order to exist and to maintain camp, the subjects usually have to perform a great deal of physical exercise such as shoveling snow, cutting wood, and the like. In fact, in many of the field studies designed to measure physiologic changes due to cold exposure, one has actually been measuring the combined effects of cold exposure and physical conditioning.

The significance of the subject's state of physical fitness in connection with tolerance to cold was emphasized by Adolph and Molnar[15] as early as 1946. In fact, many of the physiologic changes which occur in physical conditioning and training may incorrectly have been interpreted as evidence of acclimatization to cold. Physical training causes an increase in muscle mass and metabolically active body mass, as well as an increased vascularization of muscle tissues.[16] There is also an increase in

blood volume, and the total blood flow is increased by the greater cardiac output.

Experimental evidence has been reported indicating increased blood volume, rise in cardiac output,[6] and increased vascularization of muscle tissues in cold-exposed animals.[9]

3. *Energy Metabolism*—If the severity of the cold exposure of the furred or clothed organism can be measured to some degree by the increase in metabolism required to maintain life, we would expect the Eskimo's metabolism to rise to levels at least twice that of the basal, if the degree of his cold exposure were equal to that of a rat exposed to 5°C (41°F).

It is well known that the so-called "basal metabolism" of Eskimos, examined for the first time while living on their native diet, is considerably higher than that of whites. This has in the past been interpreted by several workers as indicating racial or climatic differences in basal metabolism. An extensive study of the energy metabolism of the Eskimos in Alaska[14] revealed that approximately 9% of this higher basal metabolic rate could be accounted for by apprehension, and the high-protein Eskimo diet accounted for approximately 15%. When these two factors were eliminated, the metabolism reading was almost exactly the same as in the white controls. These studies thus provide no evidence of increased basal metabolism in the Eskimo due to acclimatization to cold.

4. *Thyroid Activity*—It has been suggested that the thyroid gland is involved in human acclimatization to cold.[17] Ingbar et al.[18] observed changes, in five men continuously exposed for 12 days to 13°C (55.4°F) while wearing only cotton underwear, which were interpreted as indicating "the ability of environmental factors to modify bodily requirements for thyroid hormone" in man.

On the basis of a series of I^{131} studies on normal Eskimos, Indians, and infantry soldiers in Alaska, however, it was concluded[36] that the thyroid does not play any significant role in human acclimatization to Arctic environment when the cold stress is no greater than is normally encountered by Alaskan Eskimos or Indians in the course of their normal life or activities or by soldiers engaged in the usual Arctic service.

5. *Nutritional Factors*—On the basis of the previous statements, it would appear that Eskimos or whites living in the Arctic are not exposed to sufficient cold to necessitate the marked elevation of metabolism observed in rats exposed to 5°C (41°F). Without this increased metabolism there should be no need for increased food intake.

A dietary survey with individual food weighings for weekly periods during winter and summer in four widely separated Eskimo villages in Alaska showed that in the adult male Eskimo an average daily gross consumption of approximately 3,100 calories was sufficient to maintain the body weight with an estimated daily energy expenditure of roughly 2,700 calories throughout the year.[19] There was no appreciable difference in the food intake between winter and summer (Table I). These figures have been verified by more recent observations by Drury,[20] who found a mean daily calorie intake of 3,000 calories in two other villages. These findings are also in agreement with studies among Eskimos in Greenland,[21] where the calorie intake is in the order of 2,800 for adult males, with an average daily

Table I—Average Consumption in Male Eskimos at Four Localities in Alaska

	Calories	Protein, Gm.	Fat, Gm.	Carbohydrate, Gm.	Calcium, Mg.	Phosphorus, Mg.	Iron, Mg.	Copper, Mg.	Vitamin A, I.U.	Thiamine, Mg.	Riboflavin, Mg.	Nicotinic Acid, Mg.	Ascorbic Acid, Mg.
Barter Island													
Winter	3,800	160	164	418	980	1,800	19	2.6	3,445	1.2	1.6	23	22
Summer	3,700	157	176	380	930	2,064	24	2.2	3,388	2.0	2.0	34	34
Anaktuvuk Pass													
Summer	4,650	199	257	357	352	1,912	46	3.9	289	2.0	3.0	39	3
Kotzebue													
Winter	2,780	140	114	297	860	1,776	15	1.8	3,686	0.9	1.6	19	54
Summer	2,600	138	92	285	815	1,665	17	1.2	1,125	4.0	2.0	25	36
Gambell													
Winter	1,970	128	75	200	500	1,560	21	2.4	69,576	1.0	3.0	31	23
Summer	2,200	113	98	214	254	1,227	14	2.6	1,786	1.0	2.0	20	26
Mean	3,100	148	139	307	670	1,715	22	2.4	11,890	1.7	2.2	27	28

Table II—Daily Intake of Essential Nutrients by Infantrymen and Airmen in Alaska

Group	Calories	Water, Gm.	Protein, Gm.	Fat, Gm.	Total Carbohydrate, Gm.	Calcium, Mg.	Phosphorus, Mg.	Iron, Mg.	Copper, Mg.	Vitamin A, I.U.	Thiamine, Mg.	Riboflavin, Mg.	Nicotinic Acid, Mg.	Ascorbic Acid, Mg.
Air Force														
I October	3,500	1,309	111	147	432	1,115	1,374	17.6	2.3	6,748	1.8	2.3	20.0	32
II January	3,000	1,066	90	149	325	809	1,330	13.8	3.3	5,958	1.4	1.7	19.5	70
III April	2,400	874	76	101	299	570	1,135	14.0	3.1	7,069	2.7	2.5	18.2	80
IV July	2,900	1,491	91	107	342	1,191	1,626	15.0	1.7	5,530	2.8	2.6	19.6	113
Mean	2,950	1,185	92	126	350	921	1,366	15.1	2.6	6,326	2.2	2.3	19.3	74
Infantrymen														
I October	3,300	1,070	102	129	442	789	1,416	18.6	2.5	4,296	2.0	1.8	19.3	99
II January	3,400	1,123	103	139	432	1,156	1,751	19.5	3.3	9,280	2.1	3.0	20.7	88
III May	3,200	1,309	122	126	412	1,246	1,735	16.8	2.3	4,937	2.4	2.6	18.4	91
IV August	3,100	1,365	108	118	345	1,004	1,598	18.0	1.7	6,270	2.0	2.2	21.4	128
Mean	3,250	1,217	109	128	408	1,048	1,625	18.2	2.5	6,195	2.2	2.4	20.0	102

energy expenditure of about 2,700 to 2,900 calories.

In a group of infantry soldiers in Alaska, the gross consumption of calories varied between 3,100 and 3,400 per man per day (Table II), with an average estimated energy expenditure of about 2,800 calories. Under these conditions there was an average weight gain of 0.4 pound during the whole year. There was a slightly higher intake in winter than in summer, within a variation of 200 calories.[19] This is no more than would be expected, owing to the hobbling effect of Arctic winter clothing[22] and the greater energy expenditure required by movement through snow, etc. On the whole, these figures are no higher than those reported for soldiers stationed in a temperate climate.

In a group of airmen on active duty in Alaska,[19] the mean gross consumption was 2,950 calories per man per day. If 5% is deducted from the gross intake for unabsorbed wastage, the figure for the average net calorie consumption throughout the year is approximately 2,800 calories per man per day. The estimated mean energy expenditure was 2,700 calories. On this regimen, the body weight of all Air Force subjects for all four periods of the year remained unaltered.

These figures are in agreement with those reported in studies carried out among trappers in Greenland,[23] who maintained their body weight on an average gross consumption of 3,000 calories per man per day, varying from 3,300 in the summer to 2,100 in the middle of winter. These observations are also in close agreement with the revised National Research Council's recommended allowances of 3,000 calories per day for a physically active man of 70 kg.

The percentages of calories furnished by protein, fat, and carbohydrate in the diet of American troops living in Alaska is not significantly different from that reported for United States troops eating a garrison ration in temperate or tropical climates.[19] In airmen, however, there was an increased consumption of fat in the winter.

Europeans living in the Arctic have a very high fat intake.[23] The Alaskan Eskimo diet is a high-protein diet, but the fat intake is not very much higher than in soldiers living in Alaska (37.5% of the calories in whites, 35% to 41% in Alaskan Eskimos).

It may be concluded on the basis of these data that, contrary to conditions in the acclimatized rats, there is no evidence of increased food intake in the Eskimo which can be attributed to cold exposure or cold acclimatization. The same is true of soldiers stationed in Alaska and of trappers in Greenland.

Mitchell et al.[24] have claimed that dietary modifications exert considerable effect upon the ability of man to withstand exposure to intense cold. According to these authors, high-carbohydrate and particularly high-fat foods are to be preferred to high-protein foods and that a high-fat diet is probably superior to a high-carbohydrate diet in maintaining general psychomotor performance and visual efficiency as measured by flicker-fusion frequency.

Keeton et al.[25] have claimed superiority for a high-carbohydrate diet over a high-protein

diet in increasing the tolerance to cold in adult males under conditions in which the calorie requirements are just satisfied.

In a cold environment, however, it would appear that the heat of the specific dynamic action of protein would contribute to the maintenance of body temperature.[26, 27] The specific dynamic action of the high-protein Eskimo diet caused a 15% higher heat production in the Eskimo.[14] Glickman et al.[28] found that the total specific dynamic action after meals containing about 1,000 calories was about 169 calories for the high-protein meals (37% of calories from protein) and 103 calories for the high-carbohydrate meals (7% of calories from protein), or 17.0% and 9.6% respectively of the total calories consumed. They conclude, on the basis of their studies, that high-protein meals as compared with low-protein meals exert an inappreciable effect in protecting man against a cold environment.

Available data based on animal experimentation indicate that requirements for a number of nutrients are markedly increased under conditions of low environmental temperature. An increased requirement for thiamine and ascorbic acid has been demonstrated in animals following prolonged exposure to low environmental temperatures.[29, 30] Some animal experiments have also indicated increased requirements for vitamin A.[30, 31]

In man, on the other hand, the available experimental data have failed to demonstrate clearly any increased vitamin requirement at low environmental temperatures, nor has a beneficial effect from increased vitamin dosage in man exposed to environmental stress been convincingly demonstrated.[32]

Glickman et al.[33] studied the effect of supplements of water-soluble vitamins on the ability of 12 healthy men to withstand repeated exposures, of eight hours' duration, to intense cold (-20°F). They concluded that this experiment indicated clearly that the ability of men to withstand the damaging effects of repeated exposures to cooling environments and to maintain normal neuromuscular and mental efficiency cannot be appreciably enhanced by giving excessive doses of ascorbic acid, thiamine, riboflavin, and nicotinic acid, above the amounts required for adequate nutrition.

A carefully conducted field study with 2,500 soldiers in Sweden, however, indicated that three months of dosing with ascorbic acid produced large changes in blood and urine levels, but no changes in performance or in any of the numerous aspects of health which were evaluated.[34]

Very little is known of the actual vitamin requirements of man under Arctic conditions. In the case of ascorbic acid, the available data seem to indicate that man may remain free of deficiency symptoms on doses of ascorbic acid considerably below the figures usually considered as normal requirements in temperate climates. Man has been known to remain in excellent health in the Arctic for long periods on 15 mg or less of ascorbic acid daily.[19] In the European trappers in Greenland, the intake was less than 10 mg daily in the middle of the winter. The Greenland Eskimo's ascorbic acid consumption varied between 3 and 50 mg daily. It is also known that man may prevent and even cure scurvy on a diet of nothing but meat. This is a remarkable fact, for it would be quite impossible for a white man to obtain from meat alone the amount of ascorbic acid commonly considered as the minimum requirement. In order to obtain 30 mg of ascorbic acid, it would be necessary to consume 3 to 6 pounds of meat per day if the ascorbic acid content were 1 to 2 mg per 100 gm of meat.

Possible explanations may be that in reality the ascorbic acid requirement is less than 30 mg per day under Arctic conditions; that the content of antiscorbutic substance in meat is higher than is indicated by standard methods of assay; that a diet of meat causes changes in the intestinal flora, resulting in decreased bacterial destruction or improved absorption of ascorbic acid; and finally, that synthesis of vitamin C (ascorbic acid) may occur in man, at least to a limited extent.

In summing up our comparison between the Eskimo habitually exposed to the Arctic environment and the cold-exposed rat, we are forced to conclude that the Eskimo, after all, is simply a human being who, in response to his environment, behaves physiologically quite differently from a rat at 5°C (41°F). If we were to explore this problem of human acclimatization further, we would have to expose our subjects to far severer cold—i.e., to conditions which deviate noticeably from any normal human environment. The response might then no longer be physiological but pathological in nature, so that we might end up studying hypothermia rather than acclimatization.

In any case, it may be concluded that there is little basis for the textbook statement[35] that people who live in cold regions have an increased metabolism and that they therefore consume more food. The statement that they voluntarily increase the amount of fat in the diet may be correct in some cases, but not as a general rule.[19, 23]

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The Toxicology of Organometallic Compounds—Part I

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THE SCIENCE of chemistry has been divided classically into the areas of inorganic and organic compounds. Information on the toxicology of inorganic compounds is rather well developed in comparison to the amount of similar information available on organic substances. There are many reasons why this should be so. There are fewer inorganic compounds. Dissociation of these materials within the body is productive of ions, whose metabolism has been studied (and, in some instances, understood). The vast number of organic compounds, the difficulty in following their metabolism, and the apparent difficulty in establishing generalizations about classes, structures, or types of action of related compounds have all combined to produce uncharted areas in the field of organic chemical toxicology.

There is another class of materials about which information is equally lacking. This is the groups of hybrid offspring from the mating of organic chemicals and metals—the organometallic compounds. Materials of this class are finding increasing use in industry. As certain of these (tetraethyl lead, organoarsenic, organotin and organomercury compounds) have become of commercial interest, their metabolism and toxicological actions have been studied.

Being part metal and part organic in structure, some organometallic compounds may be expected to behave like either or both parents, and some like neither. The purpose of this review is to discuss general characteristics of this class of compounds and the toxicological information available on them in groups according to the periodic table.

The following discussions will be limited to those compounds in which there exists a carbon to metal bond (–C–Metal). This will exclude many compounds in which metals are attached to organic radicals by an oxygen atom (–C–O–Metal).

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General Properties

The properties of an organometallic compound depend largely upon the specific metal and organic radicals involved, and especially on the nature of the chemical bond between the two. The type of bond which exists depends primarily upon the types of electronic orbitals occupied by the valence or coordination electrons of the metal; the difference in electronegativity between the metal and the carbon atom to which it is attached also plays some role. The electronegativity of an atom may be defined as the sum of its first ionization potential and electron affinity, both divided by 130. The smaller the difference between the electronegativity of the metal and the electronegativity of carbon (2.5), the more covalent or electron-sharing will be the bond between metal and carbon. When this difference equals 1.7, the bond may be considered as 50% ionic and 50% covalent in nature. As the difference increases, the ionic nature of the bond will increase.

The nature of this bond is of primary importance in determining many of the physical characteristics and the chemical reactivity of the compound. The more covalent bonded (nonpolar) compounds are volatile, and soluble in organic solvents. Compounds of organic radicals with more electropositive metals, such as sodium and potassium, produce primarily ionic compounds, which are less volatile.

Period	Group I a	Group I b	Group II a	Group II b	Group III a	Group III b	Group IV a	Group IV b	Group V a	Group V b	Group VI a	Group VI b	Group VII a	Group VII b	Group VIII a
1	H														
2	Li		Be		B		C		N		O		F		
3	Na		Mg		Al		Si		P		S		Cl		
4	K	Cu	Ca	Zn	Sc	Ga	Ti	Ge	V	As	Cr	Se	Mn	Br	Fe Co Ni
5	Rb	Ag	Sr	Cd	Y	In	Zr	Sn	Nb	Sb	Mo	Te	Tc	I	Ru Rh Pd
6	Cs	Au	Ba	Hg	La (rare earths)	Tl	Hf	Pb	Ta	Bi	W	Po	Re	At	Os Ir Pt
7	Fr		Ra		Ac (series)										

Figure 1—Periodic table of elements.

The Effect of Varying Organic Radicals

In general, as the size of the organic radical increases so does the boiling point of the material. The substitution of an organic radical with branched side-chains (isopropyl, isobutyl)

(–C–C–Metal) will in general increase the volatility of the compound above that found in the normal (unbranched) radical (–C–C–C–Metal) with the same number of carbon atoms.[1]

The Effect of Varying Metals Within the Same Periodic Group

Except in Groups I and II of the periodic table, an increase in the weight of the metal *(with the same organic radicals)* produces an increase in the boiling point of a substance.

Generalizations With Regard to Chemical Reactivity

Among the metals comprising Periods Ia, IIa, and IIIa in the periodic table, (Fig. 1) there is an *increase in reactivity* as the atomic weight of the metal in the compound increases. In Groups Ib, IIb, and IIIb an increase in atomic weight of the metallic constituent, however, produces a *decrease in reactivity.* All of the members of Ia are more reactive than Ib and IIa more reactive than IIb. The above statements apply, of course, only when the organic radicals to which the individual metals are attached are the same.

In Periods V, VI, and VII, chemical reactivity decreases with increasing atomic weight.

A compound with two different radicals attached to it is usually more reactive than the same metal attached to two or three identical organic radicals[2] (R–Metal more reactive than R–Metal).

Group I Compounds—The Alkali Metals

The compounds of this group are, in general, colorless liquids and have marked chemical reactivity. Upon contact with air (or water), they will decompose with sufficient violence to either explode or burst into flame. Both the aryl and the alkyl (the aromatic and aliphatic) compounds of these metals must be handled in an inert atmosphere. Only the lithium aryl compounds demonstrate any properties of covalent bonding.

Highly reactive chemicals present a threefold problem to those who are concerned with the health and welfare of workers who handle them: first, the toxicity of the compound itself; second, the hazards associated with fire and explosions resulting from their spontaneous decomposition; and third, the toxic effects of the products of decomposition.

Group II Metals—Organometallic Compounds

Compounds belonging to this classification demonstrate a wide range of properties and reactivities due to the fact that the ionic nature of the carbon-metal bond may vary from 35% in barium compounds to a low of 11% with mercury compounds. Almost all except the organic mercurials are oxidized readily in air; many are spontaneously inflammable.

Compounds of barium, strontium, and cal-

cium resemble those of sodium and potassium. They are too reactive to be handled except in inert atmospheres. The compounds of both magnesium and beryllium are spontaneously inflammable and hydrolyze with explosive force on contact with water.

The zinc-dialkyls fume strongly and may incandesce in air. Cadmium organometallic compounds have a relative lower reactivity. They are generally liquids of moderate volatility. All in the dialkyl-cadmium series above dimethyl cadmium decompose slowly at room temperature, yielding cadmium and the alkyl or aryl components. In this case, as with many unstable organometallic compounds, the products of decomposition may be of toxicity equal to or greater than the original compound.

Organic mercurials have had wide use in industry and as drugs. They have been used by physicians in the form of diuretics. They are the most stable of the Group II organometallic compounds. The dialkyl mercury compounds are usually volatile liquids. The diaryls are solids. The alkyl or aryl mercury halides are soluble in organic solvents, and to a lesser extent in water.

Owing to the binding of mercury by the organic radicals, the metabolism of mercury contained in these compounds is quite different from that of inorganic mercury compounds. The mercuric ion binds firmly with proteins. Organic mercury compounds are more widely distributed in the body than inorganic mercury.

Organic mercuric compounds, especially diethyl mercury, have caused degenerative lesions of the central nervous system.[3] These are severest in the cerebellum.[4] These relatively nonpolar substances have a high lipid/water solubility ratio. This permits the penetration of mercury into cells under the protective mantle of its organic radicals. During metabolism of the compound, inorganic mercury may be released and have its disastrous effect upon enzyme systems and cells.

Skin absorption of organic mercury compounds is greater than that of inorganic mercury compounds. The process of skin absorption of organic mercurials may result in irritation and blistering of the skin. The acute effect of organic mercury compounds is a depression and paralysis of the cerebral centers.

Despite the difference in metabolism of inorganic and organic mercury compounds, the pictures of *chronic* intoxication from them are not too dissimilar. In the latter case, there is more degeneration in the cerebellum and the neurological signs are more pronounced.

The following hypothetical case history has been synthesized from several actual case reports. Similar "synthetic cases" are presented in subsequent sections of this paper in order to emphasize the commonest physical signs, symptoms, and course of the illnesses resulting from intoxication of a severe nature due to certain organometallic compounds.

CASE 1—A 30-year-old man received recurrent exposures to dimethyl mercury. His first complaints were increased salivation, a metallic taste in his mouth, and fatigue. One week later he noted unsteadiness of his legs, slurred speech, and generalized headache. His wife had also observed a change in his personality (depressed and withdrawn). These complaints were soon followed by the development of numbness in his fingers, toes and lips, and incoordination of arm and leg movements. Laboratory examination of a urine specimen revealed 1 to 2 + proteinuria and occasional white and red blood cells per high power field. He lapsed into coma and died about six weeks after the onset of his symptoms.

Organometallic Compounds of Group III

Most of the trialkyl and triaryl compounds of boron, aluminum, gallium, and indium inflame and decompose in the presence of oxygen. They are less polar and more covalent than Group II compounds. They are powerful electron acceptors. They are, therefore, among the most reactive of all the organometallic compounds. This is true with respect to both their vigor of oxidation and the variety of reactions which they undergo. The lower alkyls are usually colorless liquids. The aryls are crystalline solids. Of toxicological interest in this group are the dialkyl thallium halide compounds. These are stable on exposure to oxygen and water. They are crystalline solids, soluble in water and to some extent soluble in organic solvents. It is rather interesting that perhaps the most toxic of the metals in this group should form the most stable compounds with which man can come in contact. It might be expected that this class of organothallium compounds, about which there is no information available, would be extremely toxic and demonstrate (in common with their inorganic confreres) a predilection for producing injury to the central nervous system.[5]

The Organometallic Compounds of Group IV

The four elements of major interest in this group are silicon, germanium, tin, and lead. Only a few of their compounds are not tetravalent. Two of this group, tin and lead, form organometallic compounds which are of considerable commercial interest. For this reason, more is known about their toxicological actions.

The alkyl and aryl compounds of all four of these elements are relatively stable with respect to oxidation and hydrolysis. The tetra-alkyls are usually volatile liquids; the tetra-aryl compounds are usually solids.

Lead—Tetraethyl lead is an oily, volatile liquid, insoluble in water and soluble in alcohol and lipid solvents. It is well absorbed through the skin. Application to the skin is productive of some mild local irritation. In contrast to inorganic lead salts, it is rapidly cleared from the blood and distributed to a variety of tissues. While blood lead determinations are useful in the evaluation of inorganic lead exposures, they do not provide such help in those exposed to organic lead compounds. The clinical syndromes of intoxication produced by inorganic and organic lead compounds are not identical. With the former (in adults) gastrointestinal symptoms predominate and are followed by some neuromuscular disorders. Intoxication with tetraethyl lead produces vague symptoms of psychic derangement including irritability and personality changes. Some gastrointestinal symptoms may be manifest, but pallor, tremor, delirium, and convulsions usually supervene. A decrease in erythropoiesis follows intoxication with either class of compounds.

CASE 2—A middle-aged man first complained of difficulty in sleeping, nightmares, and a decrease in appetite. About two weeks later he developed a headache, diarrhea, and nausea, and his wife reported that he seemed extremely irritable. He gave a history of having been exposed to tetraethyl lead while cleaning some storage tanks for several weeks prior to the onset of his symptoms. Physical examination revealed a mild, generalized muscle weakness, coarse tremors of the extremities, and increased activity of the deep tendon reflexes. He appeared somewhat pale, had a blood pressure of 90/60, and a pulse rate of 50/minute. Results of an analysis of his urine, a white blood cell count, peripheral blood smear, and hemoglobin determination were within limits of normal. The patient required hospitalization because of maniacal outbursts. He recovered completely over a three-month period. Weakness of extensor muscles, colic, or blue coloration of gingival margins were not noted during his illness.

Tin—Inorganic tin compounds show a rather moderate degree of acute toxicity. After injection of soluble tin salts, tin is rapidly cleared from the blood. Considerable accumulation of tin occurs in the spleen and the liver.[6] Excretion is primarily via the urine and is slow. In contrast, the organic tin compounds, such as tetraethyl tin, are found to have a wider distribution and to be eliminated in breath, urine, and to a lesser extent in the feces. The lungs, blood, and adrenals are found to have the largest concentration of the metal following the administration of organic tin compounds.

There have been numerous studies of the relative toxicity of alkyl tin compounds as a result of their increased use of plasticizers and fungicides. The compounds considered in these studies fall in two general categories—the *dialkyl* tin salts, such as diethyl tin dichloride, and the *trialkyl* tin salts. The former group finds greatest use as plastics stabilizers (especially dibutyl tin salts). The latter compounds show marked fungicidal activity, up to the trihexyl compounds.

The clinical use of diethyl tin diiodide for the treatment of furunculosis on the continent produced over 100 deaths due to acute cerebral edema. Some survivors had paraplegia and other evidences of central nervous system damage.[7]

CASE 3—A 22-year-old man gave a history of having ingested a number of capsules containing organic tin compounds about two weeks prior to the onset of a severe headache. During the following week he had nausea, vomiting, and palpitations. He was admitted to the hospital in a confused state and found to have bilateral papilledema and generalized weakness, but no localizing neurological signs. Results of urine and complete blood count examinations were normal. He subsequently developed paraplegia and retention of urine, became comatose, and died three weeks after the onset of his symptoms.

Diethyl tin dichloride inhibited alpha keto acid oxidases in vitro.[8] Dimercaprol (BAL) was an effective antidote in experimental diethyl tin poisoning but was not effective against triethyl tin.[11]

The oral administration of dibutyl tin dichloride produced gastric dilatation and an inflammatory lesion of the bile ducts in rats at an oral dose of 20 mg/kg. Application of 10 mg/kg to the skin also produced lesions of the bile duct and skin injury at the site of contact. Intraperitoneal injection of 7.5 mg/kg to rats produced symptoms of shock followed by death. The same compound given orally to rabbits at a level of 50 mg/kg produced death, but without any significant autopsy changes.[9]

The relative toxicity of the dialkyl dichlorotin salts as administered intravenously to rats was as follows: The LD_{50} for dimethyl was 40 mg/kg; for diethyl, 25 mg/kg; for dipropyl, 7 mg/kg; for diisopropyl, 15 mg/kg; and for dibutyl, 5 mg/kg. The LD_{50} of di-2-ethylhexyl tin dichloride was 5 mg/kg. It would seem that increasing the size of the organic radical increases the toxicity of dialkyl tin salts. An inflammatory lesion of the bile ducts was observed, especially following administration of

dibutyl tin compounds. It was also observed in animals receiving dipropyl, diisopropyl, and dipentyl salts. With these the injury is less severe. Salts with aliphatic chains with a length of less than 3 carbons or greater than 6 did not produce the bile duct lesion. The oral toxicity of this same series of compounds was approximately the same.[9]

The acute oral toxicity of the trialkyl tin salts in rats indicated that they are relatively *more* toxic by this route than the dialkyl salts. Acute intoxication produced anesthesia. After a period during which the animals seemed to recover, they became progressively weaker and died. Autopsy findings were limited to cerebral edema, which was most marked in the white matter.[9]

The percutaneous application of tributyl tin chloride caused erythema and swelling of the follicle orifices. There are reports of skin burns with this material from contact with vapor in the clothing.[10] The mechanism of action of trialkyl tin salts is unknown. Certain studies have reported the appearance of "red tears" following injection of trimethyl and triethyl tin compounds into rats.[11] The secretion of this red porphyrin complex from the Harderian glands of rats has been shown to be the result of parasympathetic nervous system stimulation. Triethyl tin in vitro reduced the pseudocholinesterase activity of horse serum to 25% of normal. In rats in vivo, however, severe symptoms were present without measurable change in serum cholinesterase activity.[11]

The trialkyl tin salts are known to inhibit oxidative phosphorylation in vitro. Brain slices from animals receiving triethyl tin compounds demonstrated this phenomenon, while the majority of the tin compound was found to be present in the liver and red cells.[9]

Bis(tributyl tin) oxide, which is used in the control of slime in water systems, is evidently of relative lower toxicity than the trialkyl compounds themselves (oral LD_{50} rats = 194 mg/kg).[12]

The above information on the toxicity of alkyl tin compounds may be summarized by stating that both series of compounds are known to produce certain effects upon enzyme systems in vitro. The dialkyl tin salts produce symptoms of shock, followed by death without a definite sequence of symptoms. Certain dialkyl tin compounds produce an inflammatory lesion of the bile ducts. The trialkyl tins have been observed to produce symptoms of parasympathetic stimulation. The trialkyl salts produce anesthesia, followed by a period of weakness, with or without flaccid paralysis of the hindquarters. Death occurs, evidently due to acute cerebral edema.

[To be concluded.]

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Industrial Medical Association

THE PRESIDENT'S MESSAGE

D. JOHN LAUER, M.D.

IN THIS, my last President's Message, I should like to reiterate some of the thoughts I expressed in the first one.

The Industrial Medical Association is an organization of physicians interested in industrial medicine, and they cover a broad spectrum of industires, training, background, and understanding. What may be of keen interest to one may hold no appeal for another. We must, however, respect these broad differences of interests in each other and not permit any constriction of our horizon in this Association.

We have had an opportunity in the IMA which we have never been able to exploit fully —a sound idea—the promotion of the principles of occupational health among industrial management as well as physicians in general. By way of promoting these ideas and at the same time upgrading the existing in-plant health programs, plant medical departments have been surveyed by members of the IMA and certified through its legal offspring—the Occupational Health Institute. The direct cost of this activity has been borne by OHI.

The operations of the Occupational Health Institute provide an opportunity for industry and the medical profession to serve an enlightened self-interest, and by taking the initiative to prevent the requirement of rigid industrial medical services under the law.

During a recent trip abroad we were impressed in the several countries visited with the manner in which their laws spell out in some detail requirements for in-plant programs. Even the methods of administration are detailed. Management and the profession thus forfeit much of their initiative and flexibility.

During the past year we had hoped to push the in-plant certification program vigorously in a joint action with the American Medical Association. Just recently the Board of Trustees of that organization rejected this idea in favor of using all its energies and finances to oppose federal legislation which proposes to broaden the payment of medical care under the social security system.

The concepts of preventive health measures, such as health maintenance, health education, and environment control, in an in-plant occupational health program do lack some of the drama that more definitive kinds of medical care afford. They do lack the luster which comes with doing battle before the legislators. But, perhaps these kinds of programs have in them the seeds of action which might reduce the necessity of always defending our position.

One sometimes wonders if the real purposes of the Occupational Health Institute have been understood by our membership. May I invite you to reread the booklet explaining the OHI's functions and objectives. After you have done this, don't you believe that in this program for occupational health there are certain strong parallels to the National Safety Council and its tremendously successful program on industrial safety? That organization began about 50 years ago as an enlightened self-interest association of safety engineers with an idea which they were able to induce industry to support strongly. Are we missing our opportunities?

In raising these questions to you, we are inviting some active suggestions and ideas when we see each other in Rochester later this month.

In saying adieu, I can report that the greatest reward of the Presidency has been the opportunity to learn to know so many wonderful and able people. My thanks to the chairmen of the committees and their members, to the officers of the component societies, to the district counselors, to my fellow officers, and to the Board of Directors. I shall always cherish this year of my Presidency.

Book Reviews

INDUSTRIAL CARCINOGENS. R. E. Eckardt, M. D., Ph. D., F. A. C. P. Price, $6.50. Pp. 164, with illustrations. Grune & Stratton, Inc., 381 Fourth Ave., New York 16, N. Y., 1959.

In seeking to make an aphorism wherewith to characterize some of the differences between Art and Nature, the following couplet occurred to us, and it is perhaps fitting that it should appear in an American journal, for Whistler would, we imagine, approve of it, if he were in a mood to approve anything other than Whistler. It may be that he actually invented it and merely planted it first in Arcady and thence into the reviewer's mind. It goes:

Nature displays but conceals,
Art arranges and reveals.

It appeared to us that the couplet might not be an unfitting introduction to a review of a book on occupational carcinogenesis. For in no branch of medical science is the process of induction more irritatingly concealed, in spite of what must be early display, although recognition may be late by present-day methods, and the art of revelation by arrangement more tedious and so often misleading or unacceptable.

Revelation by arrangement is one way of describing the statistical management of data, and this is a branch of science.

The knack of recognizing concealed display is an art, and as with all forms of art truly so-called bears the hallmark of the artist. In the art of medicine we call success in this, early diagnosis. When cancer is in question, an aberration of structure or function must be sought with no inkling of the nature of the carcinogen. *Early* is a relative term and, frequently, early in time may be late in process. Early in this context probably implies not far advanced in pathogenesis. There is, of course, a measure of luck in this. It depends so much upon the location of the process, its accessibility and its velocity, upon the cellular nature of the tissue or organ involved, upon the lucky chance that a small involvement produces a large functional disturbance, and so on.

Whether one is a protagonist of a chemical theory or a mutation theory or a virus theory, or a genetic theory, no help can be enlisted from any to shed light on the likelihood of the abnormal display being due to a neoplastic process. The decision rests upon abnormality of function or morphology or both. Nor can any of the theories, as yet, offer a suggestion for inhibition, reversal or deflection of the process. Action is, so far, describable only by the term eradication.

It is a natural supposition that if one knew the causes or exciting agents of any form of cancer, one would be within striking distance of a chemotherapeutic approach to cure. An analogy with hormone replacement therapy may one day become a reality if some carcinogenic cellular defect is discovered. Increasing evidence of immunological aspects of malignant disease may lead to satisfactory serological diagnostic tests and perhaps a modification of classical serum treatment. The mutant theory offers less optimism if mutation is held to be a kind of act of the god of chance, but more hope if endogenous or exogenous agents are the mutagens.

Whether a plasma factor is involved is not clear, but it is hard to avoid a feeling that the environment is involved when normal cells take on a different form of life and there is some evidence to this effect. In the field of occupational cancer we are faced with a variety of probabilities as to causes of certain tumors observed to occur with greater than normal frequencies among certain categories of workers heavily and repeatedly exposed to them. Whether by substitution of different products, by total prevention of contact, by most meticulous control of emergence from the plant, the occupational cancer hazard can be prevented when it is known. There are at present relatively very few known occupational carcinogens, some dubious ones, and perhaps a host yet to be recognized or manufactured.

Eckardt, in the volume under review, has given us a most readable account of occupational cancer in a reasonably sized book. As a practiced and very experienced specialist in industrial medicine, he is well aware of the pitfalls of diagnosis and attribution to industrial causes. Diagnosis of what we have called early display is in some measure facilitated in the circumstances of occupational cancer because of the following characters.

First of all, there are relatively very few occupations in which a hazard of carcinogenesis exists, although there are many in which it appears that people have a higher than normal incidence of cancer. The author has, in a brief but informative historical essay, listed the industrial substances or processes which have in the last 200 years given evidence of being occupational carcinogens, and of these, the most significant are coal tar, shale and mineral oil, aromatic amines, ultraviolet radiation, ionizing radiation, x-rays, soot, chromate, arsenious oxides, asbestos, nickel refining, and a few miscellaneous substances which have, on the whole, not been above suspicion of having been contaminated with potent carcinogens.

When we consider soot, tar, shale and mineral oil, it is held that hydrocarbon constituents are those to which the undoubted skin carcinogenic action must be attributed and the likelihood is great that they contain important cocarcinogens.

In the case of radiations we cannot escape the experimentally demonstrable fact that mutagenesis is one aspect, and perhaps the most important, in the induction of malignant changes, whether in skin, bone or blood.

In the case of amines, only three primary aromatic amines can, as yet, be indicted as occupational carcinogens — 2-naphthylamine, 4-amino diphenyl, and benzidine (4,4′-diaminodiphenyl). These have been confirmed as bladder carcinogens in animals. 11-Naphthylamine, which has been suspected, has not thus far been shown to be carcinogenic in animals. The actual attack on the bladder epithelium may be due to orthohydroxy derivatives of these potent amines, which arise in the bladder from metabolic products of the originally absorbed amines.

Nickel refining by the carbonyl process has undoubtedly been associated with many cases of both ethmoid and lung cancer, but an identifiable carcinogen still awaits discovery.

Chromate is indicted as a lung carcinogen mainly on the statistical association between mortality from the disease among workers in the industry in Britain and on factory and insurance mortality data in the United States of America. The association of chromate manufacture with lung cancer has been "in the air" for about 50 years, but only very recently has the reviewer seen a pulmonary tumor which had been induced by Dr. Hueper, of Bethesda, Md., by injecting "chromite roast" intrapleurally in a rat, a tortuous way of getting at the lung.

Repeated failure to induce bronchogenic carcinoma in the lungs of laboratory animals with a variety of substances—e.g., tobacco smoke, nickel, and chromium compounds—is probably due to some basic functional difference between human and animal respiratory passages. One, at least, is the rich mucous goblet cells in man and their almost complete absence in laboratory animals.

Opinions and conclusions based upon the use of mice with a known low incidence of spontaneous pulmonary adenomata and the induction in them of an increased or an accelerated adenoma incidence after treatment with suspected pulmonary carcinogens are, somehow, always regarded with dubiety when one tries to extrapolate to man. This is not only because the tumors are different from squamous-cell tumors but because there is so much difference between results obtained by different workers in the field.

Asbestos as a carcinogen should perhaps better be regarded as the inducer of a precarcinogenic process. Workers who have developed asbestosis of long standing seem, according to some British postmortem statistics, to be liable to develop bronchogenic lung cancer. On the other hand, data from recent Canadian asbestos mining centers do not confirm this, even in cases of asbestosis.

Eckardt refers shortly to the possibility of something being carcinogenic in some kinds of asbestos which may be absent from others.

Arsenious oxide is a most unsatisfactory if, nevertheless, a rightful member of occupational carcinogens. The evidence for its carcinogenicity has come, first, from the cases of skin cancer and precancer among those who had for many years ingested As_2O_3 in Fowler's solution potassium arsenite solution), often in the treatment of psoriasis, and, second, from heavy exposure to arsenical sheep dip. Animal experiment has been most unsuccessful with As_2O_3.

More recently some suspicion has fallen on hematite (not mentioned by Eckardt) as a lung carcinogen, and the probably unrelated fact must be noted that certain organic iron complexes have produced sarcomata on subcutaneous and intramuscular injection in animals. A shadow of lung cancer liability has also fallen on silicotic fibrosis.

Secondly, the period of induction is long and varies according to the physical conditions and to the personal and working habits of workers. This is a rather vague term and really signifies "average period between commencement of exposure and first diagnostic sign of tumor formation." The first diagnostic sign may appear years after complete cessation of exposure, even when workers are kept under regular, routine scrutiny. The old idea that a heavily contaminated worker demonstrated achievement is now outmoded, but the notion lingers and Eckardt potently attacks it. Thus, a potential of cases is built up. In consequence, the employer must arrange for detection and treatment of such cases as they develop and at the earliest possible moment. This must be done apart altogether from the obvious demand for the most searching methods of prevention and protection. Eckardt has (p. 127) rather misunderstood the reviewer's statement on this matter as it applies to bladder tumors. Moreover, regular diagnostic examinations should continue even after a man has left the hazardous occupation.

Thirdly, the tumors arising from occupational exposures are pathologically indistinguishable from those arising in the ordinary incidence among the general population.

Fourthly, as is clearly indicated by Eckardt, occupational carcinogens attack epithelial tissues (skin, linings of bronchi, bladder, renal pelvis, sinuses) except in the special case of radioactive depositions in bone.

Fifthly, in given circumstances the time of development may seem to depend upon some inborn sensitivity or resistance, but one should be chary of concluding on this unless the constancy of exposure is almost certain, at least. When considering the selection of workers for particular jobs this is a matter of much importance. It seems to the writer that very few people completely escape some measure of epithelial overgrowth and perhaps small tumor formation in the skin even in everyday life and especially in the 40+ age-group. Some, however, have skins with lesions which are more or less accurately referred to as precancerous, and these people should not be accepted for any occupation which has a hazard of skin cancer. Eckardt is here very practical when he says" excluding such people from such work is not necessarily because they are more susceptible to carcinogens but because if they subsequently do develop skin cancer confusion may exist at to whether the cancer developed 'naturally' or as a result of the occupation."

In this country, it is probable that an employer

accepting such persons for such work would, in the event of a civil action, be held to have been negligent and heavily mulcted.

This brings us to the sixth point, which has a clear bearing on what has preoccupied the author a good deal. Since it has been shown that changes in human skin can be induced both by occupational exposure to the products indicated and by experimental exposure to the cyclic hydrocarbons present in them, the conclusion must be drawn that the latter are or can be the active agents in the former. This fact provides the link which justifies the statement that a compound carcinogenic to mouse skin can be so also to human skin.

In practice we must, sometimes reluctantly, accept the general proposition that if a compound is, by whatever means, shown to be carcinogenic to animals it cannot be overlooked that it *can* also be so to man. This proposition is the subject of endless and often fruitless argument, into which we will not enter here except to say that, if knowledge exists to the effect that a new material or a manufactured product is carcinogenic to animals, the employer must act *as if* it were carcinogenic to man. This is the basis of the common law plea for negligence on the part of an employer toward his workers, if he has not *(a)* informed himself of the extant knowledge on the products he uses or makes and *(b)* taken competent advice on precaution and hazard and acted upon it.

In Britain, as the reviewer has pointed out elsewhere, an employer is mulcted not because a worker has developed an occupational cancer, but because he has not been a good employer and has not properly protected his workers, whether by design of plant, personal protection, or otherwise. A dilemma sometimes arises when the defense is that the carcinogenicity of an industrial substance was unknown at the relevant time. In such a case the apportionment of human responsibility depends upon the good sense of experts and the even better sense of judges and juries.

This brief note gives little idea of the wide and valuable conspectus Dr. Eckardt has given, and we commend his book to all earnest young doctors, whether in industry or no.

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A CLINICAL INTRODUCTION TO HEART DISEASE. Crighton Bramwell, M.A., M.D., F.R.C.P. Price, not given. Pp. 218. Oxford University Press, Inc., 114 Fifth Ave., New York 11, N. Y., 1959.

Dr. Bramwell states that his objective is to present the basic principles of cardiology to the general practitioner and that his book is not intended for the cardiologist. This reviewer's appraisal, therefore, is not a critical analysis, such as might be undertaken by a cardiologist. The general principles of cardiology are presented, and physiology and clinical medicine are correlated in a concise manner.

In the chapter dealing with organic heart disease, the correlation of the pathophysiology and clinical findings is presented in a compact and highly readable manner, but the tenor throughout the book is the concern the physician should have for the heart patient and not the heart alone. The author stresses the importance of the patients' reaction to disease, whether organic, psychosomatic, or both. He says: "These patients' disabilities are very real to them, and by exposing their nature and thereby allaying anxiety, the physician is fulfilling a function, and conferring on his patient a benefit, no less substantial than by arresting the progress of some organic lesion."

The practice of medicine, states Dr. Bramwell, is composed of the triad diagnosis, treatment, and prognosis. Diagnosis, though most important to the physician is least important to the patient. The patient wants to know, "Is my condition serious?" and "When shall I be fit to work?"

To the reviewer, the chapter on angina of effort, its clinical features and differential diagnosis is noteworthy. Confusing causes of chest pain, such as cardiac anxiety neurosis, heart ache, local chest lesions, myofibrositis, flatulent dyspepsia, hiatus hernia, and anemia, frequently must be dealt with by the industrial physician, and Dr. Bramwell devotes adequate space to these problems.

The compactness of the book, its confinement to common cardiac diseases, and its emphasis on weighing the patient's psyche against his organic disease regarding return to employment recommend it to the industrial physician.

S. H. STRADLEY JR., M.D.

THE CLINICAL ASPECTS OF ARTERIOSCLEROSIS. Seymour H. Rinzler. Price, $8.75. Pp. 339. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1957.

Since arteriosclerotic disease is responsible for a large portion of disability among workers, this book should be of considerable interest to many physicians in industry. In the brief span of 300 pages, Dr. Rinzler presents an exceedingly well-documented account of our present knowledge of arteriosclerosis. Little or no new information in contained, but previously published data are well organized and reviewed. The orientation is clinical—the diagnosis and treatment of the various manifestations of the disease. Enough basic information concerning present day concepts of etiology, pathogenesis, and pathology are presented to clarify and justify the clinical methodology outlined. The bibliography is magnificent and should prove useful to both the clinician and the investigator.

—LEO WADE, M.D.

Editorials

Quit Complaining

AT MOST any gathering of physicians interested in occupational medicine, there is likely to be a lively discussion of the reprehensible aspects of irresponsible medical testimony in Workmen's Compensation matters. We can continue to wring our hands about this problem, or we can do something about it.

Examples of effective action are to be found in the policies of two widely separated medical organizations: the Lake County (Indiana) Medical Society and the Minnesota State Medical Association. Similar action can be taken elsewhere provided the physicians who have a sincere regard for integrity and the hope of maintaining the stature of the medical profession will but manifest the courage of their convictions. Procedures used by the two organizations are relatively simple and almost identical, but there is a significant difference in the leverage which can be brought to bear.

As handled within the Lake County Medical Society, matters of this kind have proved to be above the immunity sometimes extended to medical men of prominence. When a Society member feels that another member has been derelict in his testimony, he may request that the Industrial Relations Committee of the County Medical Society review the transcript of the case in question. If they agree that the testimony is of such nature as to bring the profession into ill repute, the man is cited to the Executive Committee of the Council of the County Society, where he is given an opportunity to express himself as to his future intentions, with the knowledge that continued deviation from acceptable medical concepts and practices will necessitate a hearing before the entire Council with further action to be determined at that time. To date, no one has elected to appear before the entire Council.

The Minnesota State Medical Association has a Committee on Medical Testimony, which upon receipt of a written and signed complaint from another physician, an attorney, or a judge will review transcripts of medical testimony. The Committee may then summon the physician who gave the testimony in question for a discussion of his statements. A choice of actions is then available: approval of the physician's testimony, reprimand, dismissal from the Association, and/or referral to the Board of Medical Examiners for suspension or revocation of the license to practice medicine in Minnesota.

In the first example cited the controls are essentially moral and, therefore, would be expected to be effective only when dealing with fellow members of a medical society. In the other, something which approaches formal legal action is at least held over the nonconforming physician—a threat that may be necessary to bring into line that man who is not a member of a Society representing organized medicine.

It is believed that a more general application of these concepts would prove beneficial to the entire medical profession in its relationship to the administration of the Workmen's Compensation laws. In matters of morality the degree to which one is his brother's keeper is always a nebulous thing, but when their relationships are so constantly interdependent that oneself and his brother, so to speak, are of alternating identity, then the challenge to one's deviating brother is akin to the removal of the mote from one's own eye.

—Guest editorial by H. Glenn Gardiner, M.D.

Drive Scared

NO FATAL traffic accident occurred in Pennsylvania over the first weekend in March —a record for highway safety unmatched in the state for more than a decade. Most of the roads were covered with snow and ice, but all were open to traffic during the two-day period. This safety record, in some part, must be attributed to a diminished traffic load on all roads and streets; but this was probably offset by the slippery condition of the roads. One concludes that driver safety increases with driver concern—and the sustained level of that concern—with the outcome of his trip. The politician running for office does well to "run scared." An equally wise policy for the motorist might be to "drive scared"; and, within limits, the worker at a hazardous job might do well to "work scared."

Abstracts of Current Literature

Clinical Toxicology

Causation, Prevention, and Control of Accidental Poisoning. H. Jacobziner.* J.A.M.A. 171:1769 (Nov. 28) 1959. (*New York City Department of Health)

This article reports on the activities of the New York Poison Control Center and is based upon the accumulated reports of 23,862 incidents of poisoning. Of all poisonings, 54.7% occurred in persons under 20 years of age. Most of the report deals with the investigation of the 13,069 poisonings in this category. It should be noted that solvents comprise 5.6% of this group; insecticides, 6.4%; and rodenticides, 2.8%. The balance of the cases were caused by medications, cosmetics, and household preparations. The article emphasizes the data-gathering function of the New York Poison Control Center and its role in delineating the epidemiology of accidental poisoning. —A.D.

Central Nervous System in Carbon Tetrachloride Intoxication. M. M. Cohen.* Neurology 7:238 (April) 1957. (*Division of Neurology, University of Minnesota Medical School)

Two fatal cases of carbon tetrachloride intoxication in adult males are reported. One case resulted from the ingestion of a commonly available cleaning fluid, and the second case resulted from inhalation of the same product. In addition to descriptions of the clinical course of each case, detailed reports are given of the gross and microscopic appearance of the brains. Principal neuropathologic alterations consisted of patchy pontine necrosis and demyelination, Purkinje cell damage, and cerebellar venous thromboses and hemorrhagic infarcts. In view of the history of heavy alcoholic intake in both cases, there is considerable speculation concerning the role of preexisting neuropathology, and the likelihood of synergism between ethanol and carbon tetrachloride —A.D.

A New Screening Test for Bromide Intoxication. G. H. Reye* and J. R. Joffe. Am. J. Psychiat. 116: 166 (Aug.) 1959. (*Northern State Hospital, Sedro Woolley, Wash.)

A simple qualitative test for the detection of bromide in urine is described. The method requires 3 ml. of urine and involves the conversion of bromide ion to free bromine. This is accomplished by the addition of acidified potassium bromate to the urine. The free bromine is extracted with either chloroform or toluene and, when present, produces an orange to red color in the extracting solvent. Correlation studies by the authors showed the test to be positive with serum bromide levels of as low as 80 mg%. The test is free from interference by other tranquilizing medications, but can be confused if iodides are present in the urine. Iodine produces a violet color in the extracting solvent and may be differentiated from bromine. —A.D.

Carbon Dioxide Poisoning: Report of Eight Cases with Two Deaths. H. I. Williams. Brit. M. J. 2:1012 (Oct. 25) 1958.

A laborer entered the hold of a ship that contained 5,000 bags of onions and a quantity of "jaggery" (a crude brown sugar obtained from the sap of palm trees). He collapsed, as did his immediate would-be rescuer. Four of six men, including the ship's doctor, collapsed during the activities. Two of the four men who had collapsed were dead on arrival at the local hospital. The doctor was conscious on arrival at the hospital, but the other man was unconscious. His breathing was stertorous, the pupils were nonreactive, and the pulse and blood pressure were elevated. The unconscious patient was treated with oxygen, nikethamide, and, later, paraldehyde to control the convulsions that developed. Both men recovered. Four other laborers who had entered the hold developed symptoms which included giddiness, pains in the legs, pain and dryness of the throat, and, therefore, were hospitalized for observation. An analysis of the atmosphere in the hold yielded an appreciable reaction for carbon dioxide. This gas may act as an asphyxiant and a respiratory stimulant or depressant, depending upon the concentration that is present, and the duration of the exposure. —L.H.M.

Dermatology

Zirconium Granulomas of the Glabrous Skin Following Treatment of Rhus Dermatitis. R. M. Williams and G. B. Skipworth. A.M.A. Arch. Dermat. 80:273 (Sept.) 1959.

Recent literature has contained reports of allergic granulomas in the axillae following the use of stick deodorants employing zirconium salts. This report deals with the development of similar granulomatous lesions on the forearms and neck following the use of zirconium oxide lotion in treating poisoning dermatitis. Histopathologic examination of a papular lesion on the forearm of one patient showed typical naked tubercles as part of the granulomatous reaction. These cellular aggregates were composed largely of epitheloid cells and Langhans giant cells.

Patch tests with the zirconium oxide lotion were negative. When, however, the material was applied to the skin and then the anointed skin was abraded with multiple punctures, the typical papular lesion made its appearance some weeks later. A biopsy of the induced lesion showed the same histopathologic picture of sarcoidal response as was noted in the forearm and neck lesions produced by the zirconium lotion. The authors conclude that

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the zirconium ion rather than a salt or any impurity is a specific sensitizer and the reactions observed were allergic granulomas. —D.J.B.

The Effect of Systemic Chloroquine Therapy on Actinic Keratoses. J. M. Knox* and J. D. Guin. A.M.A. Arch. Dermat. 80:347 (Sept.) 1959. (*Department of Dermatology, Baylor University College of Medicine. Houston, Texas)

Chronic actinic exposure is well known to influence premature aging of certain skins as well as to foster the development of senile keratoses. Frequently the keratotic lesions are transformed into malignant growths. Because chloroquine has been used successfully by many dermatologists in the treatment of light-sensitive dermatoses, including discoid lupus erythematosus, the authors decided to use the same method of suppressive therapy on two albino Negroes with severe actinic degenerative changes in the skin accompanied by numerous senile keratoses.

The keratoses regressed moderately in one patient and markedly in the other. The most impressive effects were observed early in the treatment regimen. Other patients with actinic changes in the skin have been similarly treated, with encouraging results, though not all patients in whom this methodology was employed showed improvement. —D.J.B.

The Role of Psychogenic Factors in Skin Diseases. A. Rostenberg Jr.* A.M.A. Arch. Dermat. 81:81 (Jan.) 1960. (*University of Illinois College of Medicine, Chicago)

A questionnaire used to develop information on the role of psychogenic factors in skin diseases is discussed. This questionnaire was sent to active members of the American Dermatological Association for a sampling of the opinions of the group concerning this problem. Of 108 questionnaires sent out, 107 were answered. The group almost unanimously expressed the belief that psychogenic factors are important in the development of any dermatologic condition. Many of the dermatologists were doubtful that psychiatric consultation was of any value in the handling of the patients. The cases which were most frequently referred for psychiatric help were neurotic excoriations, other factitial dermatoses, atopic dermatitis, neurodermatitis, trichotillomania, and alopecia areata. —D.J.B.

A Hand Cleanser Derived from Soybean. C. Stritzler.* Indust. Med & Surg. 28:420 (Sept.) 1959. (*Department of Dermatology, Downstate Medical Center, 450 Clarkson Ave., Brooklyn 3, N. Y.)

During the past two years, a new hand cleanser was used by 134 patients with various types of occupational dermatitis. The cases were listed as contact dermatoses from cinnamon, chromate, cement, and solvents (turpentine, gasoline, naphtha, and benzene), plus a group of dermatoses caused by overtreatment.

The cleanser is a cream containing colloidal lipid materials derived from soybean. The carrying vehicle is an acid mantle base. Seventy-five percent of the patients felt that the cleanser was an adequate one during treatment; 21% felt that, even though the cleanser did not affect the dermatitis during treatment. the product could not be considered adequate for the removal of greases, inks, and paints. Seven of the 134 patients were unable to use the cleanser under any circumstances. It is suggested that the cleanser be used during the active phase of the occupational dermatitis while the employee is continuing to work. —D.J.B.

Environmental Physiology

Methods of Cooling Subjects with Hyperpyrexia. C. H. Wyndham,* N. B. Strydom, H. M. Cooke, J. S. Martiz, J. F. Morrison, P. W. Fleming, and J. S. Ward. J. Appl. Physiol. 14:771 (Sept.) 1959. *(Applied Physiology Laboratory, Transvaal and Orange Free State Chamber of Mines, Johannesburg, South Africa.)

Cases of heat stroke and heat hyperpyrexia occurring in civilian and military occupations require immediate diagnosis and treatment if fatal consequences are to be prevented. To promote rapid cooling, it has been previously advocated that the victim be immersed in ice water or wrapped in wet sheets and fanned. The authors compared methods for dissipating body heat in volunteer subjects whose body temperatures had been elevated to 104°F by 40 to 50 minutes of heavy work at a 93°F wet-bulb temperature.

Rate of cooling was fastest when the body was sprayed with water at 87°F and exposed to air (90°F dry bulb and 87°F wet bulb) moving at 120 ft per minute. Cooling time from an assumed rectal temperature level of 106°F down to 101°F, was 45 minutes. Simply allowing the subject to rest in air at 70°F dry-bulb temperature, with low humidity but without forced convection, was almost as effective. Immersing the subject to the neck in cold water (58°F) was significantly slower in cooling the body than were the first two methods. Lowered thermal conductance of the skin by vasoconstriction and increased heat production by shivering appeared to be the principal factors in the slower cooling resulting from immersion. Moreover, there tended to be an after-drop of body temperature to subnormal levels which might contribute to circulatory collapse.

These results suggest, therefore, that excessive chilling in treating hyperpyrexia and heat stroke should be avoided. —D.M.

Body Temperature Responses to Exercise in Man on Restricted Food and Water Intake. F. Grande,* J. E. Monagle, E. R. Buskirk, and H. L. Taylor.

J. Appl. Physiol. 14:194 (March) 1959. (*Laboratory of Physiological Hygiene, University of Minnesota, Minneapolis, Minn.)

Deep body temperature is normally regulated within narrow limits. External-heat stress or increased heat production associated with heavy work, however, may impose a strain on regulatory mechanisms leading to heat storage and a rise in body temperature.

In this report, the authors measured the effect of water and food restriction on body-temperature regulation during one hour's walking on a treadmill (3.5 mph at 10% grade) at normal room temperature (78° and 40% relative humidity. In the control period, with water and food ad libitum the mean rise in rectal temperature in six subjects was 1.14 (degrees C). During the experimental period, daily water intake was restricted to 900 ml and daily food intake to 1,000 calories. Water restriction was discontinued after the work test on the sixth day. Food restriction was maintained for a total of 16 days. The increment in body temperature during the hour-work period rose during dehydration to progressively higher levels, reaching a peak of 2.65 degrees (C) on the sixth day. A major factor causing the rise in rectal temperature was a decline in sweat rate, which fell from a mean value of 820 gm/hr during the control period to 412 gm/hr on the sixth day. By the ninth day, three days after water had been restored, body temperature responses had returned to control levels despite continuation of the semistarvation diet.

These results indicate that the response of the sweating mechanism to a rise in body temperature is suppressed by dehydration. From a teleological viewpoint, this may be a mechanism for conserving body water in a manner analogous to that observed in the camel. —D.M.

Experimental Toxicology

The Acute Toxic Effects of Black Smoke. R. E. Pattle,* G. D. Wedd, and F. Burgess. Brit. J. Indust. Med. 16:216 (July) 1959. (*Chemical Defense Experimental Establishment, Porton Down, England)

Smoke generated by burning tetrahydronaphthalene (tetralin) was administered to rats, guinea pigs, and mice. The median dosage to death [(Ct)-D_{50}] ranged from 147,000 to 351,000 mg/min/cu meter at concentrations ranging from 714 to 1,140 mg/cu meter. Animals allowed to associate protected one another to some extent, probably owing to their fur acting as a filter. In mice and rats, the cause of death appears to be blockage of air passages; delayed deaths are unusual. In guinea pigs, hemorrhagic lesions are caused, and delayed deaths are commoner. Tetralin smoke appears to be about 10 times more toxic to mice and 25 times more toxic to guinea pigs than is sulfur dioxide as judged by $L(Ct)_{50}$'s, but this is not believed to provide any basis for assuming that smoke was any more responsible than SO_2 for deaths during the London fog of 1952. —R.E.E.

On the Metabolism of Some Aromatic Nitro Compounds by Different Species of Animal: Part III. The Toxicity of the Dinitrophenols with a Note on the Effects of High Environmental Temperatures. D. G. Harvey.* J. Pharm. & Pharmacol. 11: 462 (Aug.) 1959. (*Royal Veterinary College, London)

The toxicity of the following dinitrophenols (DNP) was studied in rats, mice, and rabbits: 2,3-DNP; 2,4-DNP; 2,5-DNP; 2,6-DNP; 3,4-DNP; 3,5-DNP. Data are tabulated on physical properties, the effect of temperature on LD_{50} values by intraperitoneal injection; elimination rates from blood; effect of the compounds on oxygen consumption; and relative efficiency of various measures to protect against the effects of high temperatures on poisoned animals. An observed pattern of chemical structure and toxic action is discussed. —R.E.E.

Influence of Surfactants on Absorption from the Colon. P. M. Lish* and J. H. Weikel Jr. Toxicol. & Appl. Pharmacol. 1:501 (Sept.) 1959. (*Mead Johnson & Co., Evansville, Ind.)

Two anionic surface-active agents, dodecyl sodium sulfate (sodium lauryl sulfate) and dioctyl sodium sulfosuccinate (DSS) greatly enhanced the absorption of the anionic dye phenol red from the rat colon; a nonionic agent, a condensation product of ethylene oxide with a hydrophobic base (Pluronic F68), did not. None of the surfactants influenced the absorption of methyl violet, a cationic dye. The enhancement of phenol red absorption by surfactants was antagonized by a ganglionic blocking agent (chlorisondamine) and by an anticholinergic drug (atropine). —R.E.E.

Action of Chronic Benzene Intoxication on the Phagocytic Activity of Rabbit Leucocytes. A. P. Volkova.* Gigi. i sanit. 24:80, 1959; Chem. Abst. 53:16395d (Sept. 10) 1959. (*I. M. Sechenov First Medical Institute, Moscow)

The phagocytic activity of rabbit leukocytes is reduced by the daily three-hour inhalation of 0.02 mg/liter benzene (6 ppm). This effect becomes apparent before other signs of intoxication are evident. —R.E.E.

Effects on Growing Animals of a Continuous Exposure to Experienced Concentrations of Nitrogen Dioxide. L. A. Ripperton* and D. R. Johnston. Am. Indust. Hyg. A. J. 20:324 (Aug.) 1959. (*University of North Carolina, School of Public Health, Chapel Hill)

In weanling rats exposed continuously for as long as six weeks to NO_2 concentrations of 0.15-0.5 ppm, no significant differences over controls were seen in the microscopic appearance of lung

or liver tissues. At five weeks, blood catalase values were significantly higher in the experimental group, but at six weeks they were higher in the control group. A significant increase in aspartic acid excretion occurred in the experimental group. These observations are regarded as real effects from exposure to NO_2 at concentrations found in polluted urban atmospheres, but no conclusions regarding their significance are drawn. —R.E.E.

Industrial Hygiene

The Toxicological Basis of Threshold Limit Values: 6. Report of Prague Symposium of International Threshold Limit Values. W. L. Ball.* Am. Indust. Hyg. A. J. 20:370 (Oct.) 1959. (*Department of National Health and Welfare of Canada, Ottawa, Ont.)

This international symposium seems to have presented a real opportunity for scientists from all of the western European countries and Russia to exchange views on a very basic part of an industrial hygiene program—the determination of maximum allowable concentration. All the usual methods of collecting information relative to the toxicity of industrial chemicals were reviewed and it was agreed that it is not possible or, indeed, desirable to standardize the methods of investigations used in the study of toxicity. In addition, the subject of maximal allowable concentrations in biological materials was discussed in relation to concentrations in the atmosphere or environment. Analysis of biological material—for example, blood, urine, and expired air—is of great importance in the diagnosis and prevention of occupational disease due to various industrial poisons, and the establishment of maximal allowable concentrations in biological material, analogous to those in air, would be of great interest.

Another section on the evaluation of methods of chemical analysis, physical methods, and methods of sampling used in industry was most interesting and pertinent. In this, the speakers recognize the need for simple, reliable methods and conceded that a high degree of precision was *not* essential. They felt that a precision of ± 20% in the region of the maximal allowable concentration was adequate.

An international group is . . . undertaking the publication of methods for determination of toxic substances in air. It is recommended that all other interested organizations collaborate to exchange information and experience. —E.C.R.

Public Health Aspects of Increasing Tetraethyl Lead Content in Motor Fuel. A report by the Advisory Committee on Tetraethyl Lead to the Surgeon General of the Public Health Service.* U.S. Department of Health, Education and Welfare, Public Health Service Publication No. 712, 1959. (*Public Health Service, Washington, D. C.)

The committee appointed by the Surgeon General reviewed the available information relative to the public health hazard from increasing the concentration of tetraethyl lead in the gasoline sold as motor fuel. The report contains interesting and useful information concerning the consumption of tetraethyl lead in motor fuel, concentrations of lead as well as oxides of nitrogen in the automobile exhaust, and a little information concerning the lead content of the air in an urban area. Although the data suggest that the contribution of the atmospheric lead resulting from tetraethyl lead in motor fuel is a small fraction of the total body burden, the committee was concerned about the inadequacy of medical data derived from human exposures on which to base a conclusive answer to the problem. If data were now available on body lead burdens, with 1926 as a baseline, a more objective decision would have been possible.

The Committee concluded that "(1) a change in the maximum concentration of tetraethyl lead in motor fuel from 3.0 to 4.0 cc would not increase the hazards involved in the manufacture and distribution of leaded gasoline and (2) available data do not indicate that such change would significantly increase the hazard to public health from air pollution." Further the Committee suggested that if the concentration is increased, three precautions be observed:

1. The increase should be gradual, not to exceed 0.25 cc of lead in any one year or 3.2 cc per gallon at the end of five years.
2. Public Health Service be supplied with domestic consumption figures for tetraethyl lead.
3. Further studies on atmospheric lead concentrations and body burden of lead for selected population groups be studied. —E.C.R.

Internal Medicine

Therapy of Diabetes Mellitus with Combinations of Drugs Given Orally. S. B. Beaser.* New England J. Med. 259:1207 (Dec. 18) 1958. (*Harvard Medical School, Boston.)

In diabetes, there are defects of carbohydrate metabolism in the liver as well as a defect in the transport of glucose into peripheral muscle. The recently introduced hypoglycemic agents are used in an attempt to correct one or more of these defects. These agents may be divided into two classes.

I. The sulfonylureas (tolbutamide and chlorpropamide): This group stimulates the pancreas and influences certain hepatic enzymes concerned with carbohydrate metabolism, resulting in decreased hepatic glycolysis and glucose output and an increase in liver glycogen.

II. The diguanides: This group decreases hepatic glucose output and increases muscle uptake.

The different actions of these groups suggested the possibility that their combined action might prove beneficial.

Patients chosen for this study had diabetes of more than two years' duration, which could not be regulated by diet alone, and their insulin could not be replaced successfully with either tolbutamide or carbutamide alone.

In this limited study, it appears that a combination of phenethyldiguanide with tolbutamide or chlorpropamide has an additive or even a synergistic effect. The detailed results in four patients are reported. Two patients were able to replace insulin with this combined therapy. No toxic effects were noted. —S.H.S.

The Diagnosis of Prepsychotic Schizophrenia. A. Meares.* Lancet 1:55 (Jan. 10) 1959. (*Melbourne, Australia.)

Schizophrenia develops only in persons of introvert personality. Some introverts overcompensate and present a picture of extroversion. These individuals are pseudoextravert, and it is important to recognize them. The introvert is a highly sensitive person. He defends himself by withdrawal, which eventually, if the defense is pushed too far, merges into the apathy of schizophrenia. A person with fully developed schizophrenia is relatively free of anxiety, but in a young introvert anxiety is suspect of prepsychotic schizophrenia. The anxiety is expressed as loss of contact with the outside world or homosexual conflict. A diagnostic point also is a sudden mild euphoria associated with greater withdrawal and increased interest in the abstract, in contrast to improvement in an introvert, where a happier state of mind is associated with greater socialization and firmer contact with reality.

The author discusses symbolic thinking, literal thinking, predelusional thinking, devaluation of reality, revaluation of basic biological events and religious and political beliefs. Types of behavior such as increased introversion, decreased introversion, lack of propiety, fads, and mannerisms are discussed.

The diagnosis of prepsychotic schizophrenia requires careful evaluation of the individual's thought and behavior. Change in the magnitude of a particular sign is of greater importance than the sign itself. —S.H.S.

Asian Influenza—Clinical Picture. G. E. Burch,* J. J. Walsh, and W. J. Mogabgab. A.M.A. Arch. Int. Med. 103:696 (May) 1959. (*Department of Medicine, Tulane University School of Medicine, 1430 Tulane Avenue, New Orleans 12)

An epidemic of Asian influenza in the summer and fall of 1957 permitted a careful clinical study of some 76 "proven cases" of the disease. Clinical disease plus virus recovery and/or a fourfold or greater increase in hemagglutination inhibition antibody (HIA) titer was required for inclusion of any case in the study. The patients were primarily young adults, about equally divided as to race and sex. Numerous symptoms were observed, but fever, chills, myalgia, headache, and cough were most frequent, but by no means constant. Nasopharyngeal and conjunctival injection was seen in a third to a half of the cases, but pulmonary signs were infrequent (10% or less).

The disease was usually mild to moderate in severity, but the existence of preexisting disease, such as cardiac disease, or the presence of pregnancy seemed to aggravate the infectious process. —L.W.

Medical Care Administration

Implications of Blue Cross Hearings for Hospitals. R. A. Nelson.* Hospitals 33:31 (April) 1959. (*Director, Johns Hopkins Hospital, Baltimore, Md.)

The majority of Blue Cross plans operate under state laws that require some sort of approval by the insurance department. State regulations cover approval of contracts—therefore, rates of payment by Blue Cross to hospitals for hospital care given. For many years insurance department hearings on Blue Cross were routine affairs with little public interest or concern about hospital operations. In the past two years this situation has changed markedly. The scope of the hearings has been expanded to include examination of hospital service and medical practice as well as a review of the efficiency and soundness of the financial side of Blue Cross operations. The public is demanding more and better hospital care but wants to be sure it is not paying too much for it.

The Baltimore, Philadelphia, and New York City hearings followed a similar pattern. The question of Blue Cross-hospital relationship came in for a liberal airing. The so-called abuse of hospital care by Blue Cross subscribers was one of the major issues discussed. The hearings also brought out that acute hospital care is meeting only part of the public's need. Prepaid benefits must be extended to the walking patient.

The major implications for hospitals of the public hearings is that more controls are in the offing. These will come about through public pressure to keep Blue Cross rates from increasing. Hospitals are confronted with the prospect of imposing these controls on themselves —A.B.

Comparison of Prematurity and Perinatal Mortality in a General Population and in the Population of a Prepaid Group Practice, Medical Care Plan. S. Shapiro,* L. Weiner, and P. M. Densen. Am. J. Pub. Health 48:170 (Feb.) 1958. (*Division of Research and Statistics, Health Insurance Plan of Greater New York)

This report deals with the pregnancy experience of members of the Health Insurance Plan (HIP) as compared with the general population of New York City. The study was undertaken jointly by HIP and the New York City Health Department.

A higher proportion of women in HIP began their prenatal care in the first trimester. The prematurity rates were lower in HIP. The perinatal mortality rate for New York City was 38.1 per

1,000 live births and fetal deaths, while the corresponding figure for HIP mothers was 23.9.

A question of major importance is whether there are differentials in pregnancy loss attributable to population characteristics not yet identified or whether they are due to variation in the medical care setting of the two populations. Some consideration must be given to the socioeconomic status. The city has about 1 Board-certified obstetrician-gynecologist per 3,300 women in the child-bearing age. The corresponding ratio for HIP is 1 per 1,000.

If this more frequent use of certified specialists explains the lower perinatal mortality rate in HIP, it has great significance for community maternal and child health programs. —A.B.

Role of the Medical Society in Occupational Health. K. Davis.* Indust. Med. & Surg. 28:322 (July) 1959. (*Medical Director, Phillips Petroleum Co., Bartlesville, Okla.)

The subject is approached from the standpoint of organization, education, and liaison. There should be an active committee on occupational health in every state medical association; there are now 41. Surveys indicate that 90% of the physicians in general practice take care of industrial medical cases, three-fourths of the working population being employed in plants too small to have full-time medical directors. When we realize that all branches of medicine except pediatrics have some relationship to industrial medicine, the most important role of the medical society in occupational health is the education of professional and lay groups in the needs, benefits, ethics, and practice of this specialty. This can be done (a) by planned programs, meetings, and joint activities and (b) by fostering medical education in occupational health during undergraduate medical school years and encouraging more young physicians to enter the practice of occupational medicine.

Good occupational health is dependent upon many medical disciplines working together and jointly with management and labor. The county medical society has a responsibility to encourage liaison between its members who do industrial medical work and the various local groups and organizations (medical, industrial, civic, social welfare, and public health) which are concerned with problems of occupational health. —A.B.

Mental Health

Brief Psychiatric Approach for the Clinician. H. Winn.* J.A.M.A. 172:226 (Jan. 16) 1960. (*Comprehensive Medicine Clinic, Temple University School of Medicine, Philadelphia, Pa.)

One of the common criticisms of the evaluation and treatment of psychiatric problems is that it is too time-consuming. The author, teaching in a medical clinic, concedes this to be valid criticism and outlines a method of brief evaluation especially designed for the first interview when decisions must be made on diagnosis and therapy. This is by no means a complete method of handling emotional problems, but it is a useful suggestion for the busy physician and will appeal to the occupational physician. The first task is to understand the meaning of the disease to the patient. "One must understand that every patient has fantasies about the etiology and significance of his symptoms but may be reluctant to speak about them and occasionally even to face them consciously." An example is given with a few simple questions which may be asked to elicit the patient's attitude.

People who are emotionally ill utilize their symptoms in the service of their neurosis. As a helpful question to ask the patients, he suggests, "Would your life be very different if you did not have this condition?" The patient who is neurotic usually will answer that his life would be considerably different. For example, an obese girl overeats so that she will not have to face the stresses arising from living a more socialized life. Obesity is a defense.

The author made observations concerning the doctor-patient relationship: "First the patient must be able to communicate so that we can get an adequate history and physical examination. Second, he must be able to cooperate actively with the physician in his plan of management. Third, he must act in such a manner as to allow us to feel relatively comfortable in the practice of our profession." It is suggested that the type of approach outlined here will enable the busy physician to come to grips and assess the complex factors in the interrelationship of the patient and his disease. —G.C.T.

Insurance for Mental Illness and General Hospital Care. J.A.M.A. 172:341 (Jan. 23) 1960.

This is a condensed summary of the proceedings of the Sixth Annual Conference of Mental Health Representatives of Sate Medical Associations, held in Chicago on Nov. 20 and 21, 1959, sponsored by the American Medical Association Council on Mental Health. The 175 participants were divided into panels, and the reports of the panels are summarized. Topics include Mental Health Insurance, Oversized Institutions, More Open Treatment, Changes in Commitment, Release and After-Care, and the The Role of Private Hospitals.

Concerning mental health insurance, it is pointed out that, in many localities, psychiatric patients do not receive benefits comparable to those available for other medical conditions. It is desirable to attempt to obtain coverage for mental patients in a manner similar to that available to patients for other illnesses. Although the private psychiatric hospitals have only 2% of the beds in the United States, they receive, nevertheless, 40%

of the acutely disturbed patients on their first admissions. A plea is made for organized medicine to obtain budgetary and legislative support for the improvement of the care of the mentally ill. —G.C.T.

Noise and Hearing

Relation Between Loudness and Duration of Tonal Pulses: I. Response of Normal Ears to Pure Tones Longer than Click-Pitch Threshold. F. Miskolczy-Fodor.* J. Acoust. Soc. America 31:1128 (Aug.) 1959. (*Research Department, New York Eye & Ear Infirmary, 218 Second Ave., New York 3, N. Y.)

In normal ears, loudness decreases and audibility threshold rises when the length of a tonal pulse is shortened below a time limit, here called the "adjusting time" of the auditory mechanism. Below this time limit, intensity-duration relationships assume certain proportions. If duration is decreased by a factor of 10, the threshold rises some 8 to 10 db. The threshold intensity as a function of pulse duration was measured on 40 normal ears. For these frequencies, threshold intensity rises as the pulse duration decreases below 300 milliseconds. The mean change (rise) in thresholds as duration varied was 300 msec—0 db; 100 msec—1.55 db; 30 msec—5.87 db; 10 msec—10.44 db; and 3 msec—14.82 db. These experiments were reversed—level above threshold was fixed and a "time threshold" determined on 20 normal ears. The results are shown in these samples: 2 db above threshold—time duration =86.5 msec; 5 db above threshold—time duration =43.0 msec; 10 db above threshold—time duration =13.7 msec; 16 db above threshold—time duration =3.2 msec. The "time threshold" varies with frequency, particularly the lower frequencies. The time threshold was shown to be related to loudness level in phons and to loudness in sones. The details of these relations are given in four figures and six tables. The investigations confirmed the close relationship between duration of a tonal pulse and loudness sensation for tones which are shorter than the adjusting time of the hearing organ. —H.O.P.

Identification of Elementary Auditory Displays and the Method of Recognition Memory. I. Pollack.* J. Acoust. Soc. America 31:1126 (Aug.) 1959. (*Air Force Cambridge Research Center, Hanscom AFB, Mass.)

The fact that the listener is forced to employ a set of arbitrary designations (numbers, etc.) in identifying auditory displays has been a stumbling block in many studies on identification of elementary displays. Use of the "method of recognition memory" circumvents these difficulties. In this method, members of a defined set of objects (or sounds) —the exposure set—are initially exposed to the observer. An equal number of members outside the exposure set—the confusion set—are randomly mixed with the exposure set. Members of this combination—the combined set—are then presented, one at a time, to the observer. His task is to report which items belong to the exposure set and which items do not belong to it. The method is directly applied to identification of elementary auditory displays; six different sound levels of tone are presented and then six other levels are intermixed with them and presented one at a time. The listener reports which tone level was in the original exposure set and which was not. In these experiments, 24 different sound levels of a 1000 cps tone were used in 2.5 db steps from a sound level of 52 db to a level of 109.6 db. Special methods were developed for treating and presenting the data. An index of performance is given by a measure of discrimination called the mean ds score (index). It was found that the ds index decreases as the size of the exposure set increases. A low performance (ds=0.36), or 57% correct identification and acceptance, was given by the most proficient group of subjects when the exposure set consisted of six sound levels, or the total combined set was 12 sound levels. One often asks, "how many sounds can be identified by a listener?" If a 75% correct two-way symmetrical classification is required, then an exposure set of two or three sound levels or a combined set of about five sound levels is all that can be used. —H.O.P.

Radiation Health

Some Aspects of Mammalian Radiobiology. R. H. Mole.* Radiation Res., Supp. 1, p. 124, 1959. (*Medical Research Council, Radiobiological Research Unit, Harwell, England)

A fundamental premise is cited that one can only predict behavior of a complex organism by observing effects on the mutual interaction of cells as opposed to more detailed cellular or chemical studies. The lack of systematic relationship between data on different mammals is illustrated to show the hazard of extrapolations between species.

The observations of increased incidence of leukemias and cancers in humans following radiation exposure are briefly reviewed. These are placed in perspective by discussing the problem of assessing hazards which result only in increased frequency of events which occur "spontaneously" all the time.

It is shown that large, single radiation doses produce a steep dose-mortality curve so that relatively small radiation dose changes near the LD_{50} range correspond to large percentile mortality variation. This permits demonstration of small degrees of therapeutic effectiveness among radiation protectants. It is noted that the chemical protectants act by making naturally more-resistant animals to become more radioresistant, while damage to radiosensitive animals is little changed. This unexpected phenomenon needs explanation.

Uncertainty exists as to whether premature

aging can be considered a delayed effect of radiation exposure. It is pointed out that measuring life shortening does not measure aging per se. Description of possible relationships between dose and damage from chronic irradiation concludes that characteristic mammalian responses suggest that a balance can be reached between continued repair and continuing damage. Studies indicate that dose intensity may be a most important factor in the causation of certain types of radiation damage. —G.L.V.

Late Effects of Internally Deposited Radioisotopes in Laboratory Animals. M. P. Finkel.* Radiation Res., Supp. 1, p. 265, 1959. (*Division of Biological and Medical Research, Argonne National Laboratory, Lemont, Ill.)

The results of internal radioisotope exposures are difficult to evaluate because the total dose and dose rate cannot be controlled or adequately measured, since they continuously vary with time and space within the tissues. This summary of experiments using intravenous injection of Ra^{226}, Pu^{239}, U^{233}, Po^{210}, Sr^{90}, and Ca^{45} in mice presents a comparative analysis of late effects in terms of reduction in life span and induction of tumors. Factors influencing these effects include the type and energy of the radiations, the amount of isotope retention in the body, and the distribution of the retained material.

Comparison of the injected microcuries of these isotopes shows the α-particle emitters are more toxic than the β-particle emitters. In this group, radium is the least effective α-emitter in reducing life span. It is less effective than Pu^{239} in the production of osteogenic sarcomas but slightly more potent than U^{233}. The major factor in the increased effectiveness in bone tumor production of Sr^{90} compared with Ca^{45} is the difference in β-ray energy.

All the isotopes accelerated the appearance of tumors of the blood-forming tissues. Dose levels of Sr^{90} which were insufficient to reduce life span or produce bone tumors caused reticular tumors to occur significantly earlier than in the controls. Since these tumors bear some resemblance to human leukemias, they are important subjects for further study. —G.L.V.

Surgery

Injuries of the Kidney. C. Rieser.* Am. Surgeon 25:657 (Sept.) 1959. (*Emory University School of Medicine, Atlanta, Ga.)

A review of 137 cases of kidney injury between December, 1947, and January, 1958, is presented. The diagnosis of renal injury is made by noting the history of trauma, pain in the side, possible palpable tumor, muscle guarding, and the presence of blood in the urine. It is extremely important to ascertain the extent of injury, degree of laceration, and presence of extravasation of urine and blood.

Patients with evidence of intraperitoneal injury need not have an extensive urologic workup, since exploration reveals the magnitude of the trauma. If circumstances permit, establishing the presence and function of the uninjured kidney by excretory urography is important. An error in judgment might be committed if the decision for surgical exploration were based solely on the clinical status and excretory urography. The additional details which allow the specific decision for emergency surgery or expectant observation to be made can be furnished by cystoscopy and ascending pyelography. By thse procedures, the presence of extravasation of urine and the amount of retroperitoneal hemorrhage can be estimated.

Conservative medical observation is the method of choice only in contusions of the kidney. In this series, experience indicates that in cases of all other kidney injuries exploration should be performed after stabilization of the patient's circulatory system and if no other coexisting condition contraindicates it. Exposure permits full appraisal of the situation, and hemorrhage can be arrested, hematomas evacuated, unrecognized extravasation drained, infarcted areas resected, lacerations sutured, delayed infection minimized by drainage, and nephrectomy performed if necessary. Furthermore, the late sequelae to inadequately treated injuries can be avoided. —S.S.

Traumatic Rupture of the Spleen. B. D. Overbrook* and S. Pearson. Am. Surgeon 25:900 (Nov.) 1959. (*University of California Medical Center, Los Angeles 24, Calif.)

Seventy-three cases of traumatic rupture of the spleen, with an over-all mortality of 9.7% and an operative mortality of 5.4%, are reviewed. After abdominal trauma, a positive peritoneal tap is indication for surgery. Since one negative tap does not rule out splenic rupture, taps in each of the four quadrants may be necessary. When abdominal taps are negative, careful observation is required for delayed splenic hemorrhage. Multiple injuries decrease chances for survival. Abdominal exploration should not be withheld in cases of intraperitoneal bleeding associated with multiple injuries.

Patients should receive blood prior to surgery. Failure to respond to 2 or 3 units must mean massive and continuous bleeding, and the need for surgery is urgent. Intelligent use of blood in adequate amounts is recommended. Cyclopropane is recommended as the anesthetic of choice. With postsplenectomy platelet elevations, and increased tendency toward thrombosis may be observed. Adequate hydration is necessary, and anticoagulants may be required. Other complications are subphrenic abscess, secondary bleeding from vessels not carefully ligated, wound dehiscence, and benign splenic-flexure stricture. —S.S.

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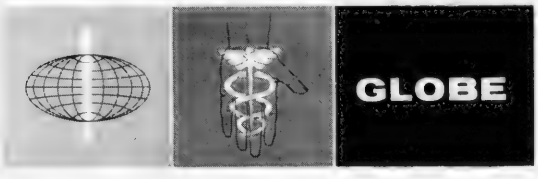

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About the Authors

S. CHARLES FRANCO, M.D., F.A.C.P., is Executive Medical Director of the Consolidated Edison Company of New York, Inc. He graduated from the Long Island College of Medicine in 1931 and completed a residency in internal medicine at the Long Island College Hospital in July, 1934. His association with the Medical Department of the Consolidated Edison Company began in July, 1935, and he joined the full-time staff of the Consolidated Edison Company in January, 1949. In World War II, he was Commanding Officer of an Army Field Hospital in the ETO.

DR. FRANCO is a Fellow of the American College of Physicians, a specialist certified by the American Board of Internal Medicine and also by the American Board of Preventive Medicine in Occupational Medicine. He holds the title of Associate Clinical Professor of Industrial Medicine at the New York University-Bellevue Medical Center. His work in the field of industrial medicine has covered such subjects as problem drinking in industry, cardiovascular disease in industry, and periodic health examinations. His article in the present issue is entitled, "A Company Program for Problem Drinking: Ten Years' Follow-Up." DR. FRANCO is a director of the Industrial Medical Association.

A. C. STIMSON is a civil engineer and surveyor by profession. He is Honorary Curator of Reptiles, Houston Museum of Natural History and the Houston Zoo. He is also a charter member of the Board of Trustees of the Houston Museum of Natural History. In addition, MR. STIMSON is a member of the Mayor's Zoological Advisory Committee and a Fellow of the Texas Association for the Advancement of Science. For the past decade, he has acted as a consultant to physicians of Houston and Harris County in several hundred cases of snakebite. Hospitals in this area call him in most instances of snakebite. MR. STIMSON has been an avid student of herpetology for more than 45 years. He is senior author of the article in this issue, "The Treatment of Snakebite."

HUGO T. ENGELHARDT, M.D. is an associate professor of clinical medicine at Baylor University College of Medicine, Houston, Texas, and an associate physician at Jefferson Davis Hospital and for 10 years was chief of the Diabetes Clinic of these two institutions. Since 1945 he was been an internist in the Medical Division, Humble Oil and Refining Company. He is co-author of the article, "The Treatment of Snakebite."

DR. ENGELHARDT, a native of Texas, attended the University of Texas at Austin, and studied medicine at Tulane University. He received the M.D. degree from Tulane in 1937. After an internship and a brief period in general practice in San Antonio he returned to Tulane, where he remained for six years, first as a fellow in internal medicine and later as a faculty member on the staff of Charity Hospital, where he became Chief of White Diabetes Clinic. His move to Houston with affiliations at Humble, Jefferson Davis Hospital, and Baylor occurred in 1945.

DR. ENGELHARDT is a Diplomate of the American Board of Internal Medicine, a Fellow of Texas Academy of Science, and a past president, Texas Diabetes Association.

From Oxford University comes the article in this issue "The Effect of Cold on Manual Performance." KENNETH ALFRED PROVINS, Ph.D., the senior author of the article, is a member of the Unit for Research on Climate and Working Efficiency in the Department of Human Anatomy at Oxford. Born in London, DR. PROVINS was educated at Wimbledon C.S. and Brasenose College, Oxford. He received the B.A. degree from Oxford with Honors in Physiology and Psychology in 1949, the M.A. degree in the same year, and the Ph.D. degree in 1958. From 1949 to 1952 DR. PROVINS undertook research in the field of human engineering for the Ministry of Supply. Since 1952

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he has been a member of the Medical Research Council Unit for Research on Climate and Working Efficiency at Oxford. His published works have been mainly on various aspects of perceptual-motor skill.

RICHARD CLARKE, M.D., who is associate author of the article "The Effect of Cold on Manual Performance," is a product of Northern Ireland. Born near Belfast, he was educated at the Royal Belfast Academical Institution and later at Queen's University of Belfast. From this university he received the B.Sc. degree with Honors in Physiology in 1951 and the M.B. degree in 1954. He worked in the Medical Research Council Unit for Research on Climate and Working Efficiency in Oxford from 1955 to 1958, mainly on the effects of local temperature on forearm flood flow and muscular performance. He received the M.D. degree in 1958. DR. CLARKE is now Senior House Officer and Registrar in Anaesthetics in Belfast.

KAARE RODAHL, M.D., Dr. med., is a Norwegian who relatively recently became firmly identified with medical research in the United States. He received the M.D. degree from Oslo University in 1935, and thereupon entered graduate studies which were temporarily interrupted by the war. He received the Dr. med. (Medical Sciences) degree in 1950 from Oslo.

At about this time, DR. RODAHL developed a consulting relationship with the Aeromedical Laboratory at Dayton, Ohio, and this was followed by responsibilities as Chief of the Department of Physiology, Arctic Aeromedical Laboratory, Ladd Air Force Base, Alaska. In 1954 DR. RODAHL left his post as Assistant Professor of Physiology at Oslo and became full-time Director of Research, Arctic Aeromedical Laboratory, Ladd AFB, Alaska. In 1957 he became Director of Research, Lankenau Hospital, Philadelphia.

DR. RODAHL has been involved in Arctic expeditions since 1937, when he was 20 years of age. He has published several books on the Arctic and a series of articles pertaining to environmental physiology and medicine, pathology, nutrition, and biochemistry. —EDITOR

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People and Events

► ROBERT T. LEGGE, M.D., one of the foremost pioneers in the field of industrial medicine died on March 21 at his home in Berkeley, California. He was 87 years old. A tribute to DR. LEGGE on his 87th birthday was published in the July, 1959, issue of the JOURNAL OF OCCUPATIONAL MEDICINE.

► CHARLES G. MERCKEL, M.D., has been appointed full-time Medical Director for the Mountain View Operations of Sylvania Electric Products, Inc., in Mountain View, Calif. He succeeds JOHN LOPES, M.D., who served Sylvania on a part-time basis and has returned to private practice.

Before joining Sylvania, DR. MERCKEL had been on the medical staff of Southern California Edison Company since 1951. He was also preferred medical examiner for the Mutual Life Insurance Company of New York.

DR. MERCKEL did his undergraduate work at Wayne University in Detroit, Mich., and later was awarded the M.S. degree on the basis of experimental work in the physiology of reproduction. He received his M.D. degree from Wayne University School of Medicine in 1940.

During World War II, DR. MERCKEL served with the U.S. Navy for four years and now holds the rank of commander in the U.S. Naval Reserve. He is a Fellow of the American Geriatrics Society and a member of the Industrial Medical Association and the American Association for Advancement of Science.

► THOMAS G. RIGNEY, M.D., formerly Senior Physician of the Standard Oil Company (New Jersey), has been appointed Acting Assistant Director of the Medical Research Division, Esso Research and Engineering Co., Linden, N.J.

DR. RIGNEY obtained his medical degree from Cornell University Medical College in 1944 and interned at Bellevue Hospital, New York City. Following service in the U.S. Army Medical Corps, he became an instructor in anatomy at Cornell University. From 1950 to 1952, DR. RIGNEY was Director, Kips Bay Cancer Detection Clinic and Director of Student Health Services, Cooper Union, New York City. He joined Standard Oil in 1953 and also served as Instructor in Public Health and Preventive Medicine at Cornell University Medical College. In 1957, he was certified as a specialist in Occupational Medicine by the American Board of Preventive Medicine.

DR. RIGNEY is a Fellow of the Industrial Medical Association and for the past several years has served as chairman of the Public Relations Committee.

► BERTRAND E. BENNISON, M.D., has joined the Ortho Research

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Foundation as Assistant Director of Research.

DR. BENNISON did his undergraduate work at Massachusetts Institute of Technology. He attended Harvard Medical School and obtained his M.D. degree in 1941. After an internship at U.S. Marine Hospital, Staten Island, N.Y., he entered the U.S. Public Health Service and spent the next 12 years at the National Institutes of Health in Bethesda, Md., doing research on malaria and cancer.

During 1953-1954, DR. BENNISON attended the University of Pittsburgh's Graduate School of Public Health, and received the M.P.H. degree. He then joined Esso Research and Engineering Co., Linden, N.J., as Chief Clinical Research Physician, and later became Assistant Director of the Medical Research Division.

DR. BENNISON is a member of the Industrial Medical Association, and a Diplomate of the American Board of Preventive Medicine certified in the specialty of Public Health, 1955.

► ROBERT S. AYERLE, M.D., has been appointed Medical Director of The Bell Telephone Company of Pennsylvania, succeeding RICHARD J. CAMPION, M.D., who has retired. DR. AYERLE joined the company in October, 1959, as Assistant Medical Director.

He received a B.S. degree in chemical engineering from Villanova University in 1942. After a year's further study in chemical engineering at Princeton University, he attended the University of Pennsylvania Medical School where he received his M.D. degree in 1951. He interned at Walter Reed Army Hospital in Washington, D.C.

DR. AYERLE served with the U.S. Army Medical Corps as First Lieutenant from 1951 to 1953. He then joined the Pennsylvania Railroad as Medical Officer, and subsequently held the position of Regional Medical Officer, later becoming Assistant Medical Director. He is a member of the Industrial Medical Association.

► The Section of Industrial Medicine and Surgery of the Los Angeles County Medical Associa-

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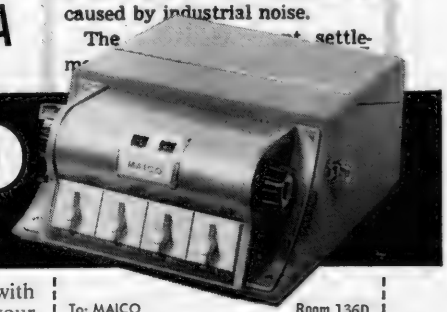

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People and Events

tion has honored A. C. REMINGTON, M.D., posthumously, for his many contributions to the field of industrial medicine. DR. REMINGTON died May 6, 1959, at the age of 44. A scroll of appreciation was presented by CLARENCE L. LLOYD, M.D., to DR. REMINGTON'S widow.

The wording on the scroll was as follows: "Section of Industrial Medicine and Surgery of the Los Angeles County Medical Association confers this scroll to A. C. REMINGTON, M.D., 1918-1959, who as Past President of this Section and Secretary of the Western Industrial Medical Association made an unforgettable and invaluable contribution to the field of Industrial Medicine. His unflagging devotion and boundless energy has provided lasting inspiration to all those with whom he has been associated in his private and professional life."

► The following physicians have accepted invitations from the Massachusetts Industrial Nurses Association to serve two-year terms as members of the group's Medical Advisory Council: GEORGE E. SPENCER, M.D.; FREDERICK G. MCKERR, M.D.; ROBERT C. THOMPSON, M.D.; RONALD F. BUCHAN, M.D.; WILLIAM F. HICKEY, M.D.; NORMAN W. WHITE, M.D.; HENRY F. HOWE, M.D.; and ROBERT L. QUIMBY, M.D.

► Northwest Association of Occupational Medicine has recently elected the following officers: W. D. NORWOOD, M.D., President; A. JOHN NELSON, M.D., Vice-President; W. J. SITTNER, M.D., Secretary-Treasurer; and CLARA MEHRER, Assistant Secretary-Treasurer. Elected to the Board of Directors were FORREST E. RIEKE, M.D.; L. A. PATTERSON, M.D.; and EUGENE P. OWEN, M.D.

►New York State Society of Industrial Medicine, Inc., held a luncheon meeting at the New York University Club in New York City on March 30. WILLIAM P. SHEPARD, M.D., Chief Medical Director, Metropolitan Life Insurance Company, and member of the American Medical Association's Council on Industrial Health, spoke on the subject of "Retirement in Industry."

► Michigan Industrial Medical Association will meet on the morning of April 8 in Midland. The program includes a tour of the facilities of the Dow Chemical Company. For further information, contact HENRY J. KREULEN, M.D., Fisher Body Plant No. 1, General Motors Corp., Grand Rapids, Mich.

► The North American Aviation Corporation has been awarded the Laca Pioneer Award by the Los Angeles Committee on Alcoholism, Inc., for the company's "demonstrated interest and participation in the community action program on alcoholism."

The Proclamation of Achievement was accepted by CLARENCE L. LLOYD, M.D., Medical Director, who praised the work of the Los Angeles Committee on Alcoholism and credited a large measure of North American's success in dealing with the problem of alcoholism to the efforts of the company's counselor, EARL SCOTT.

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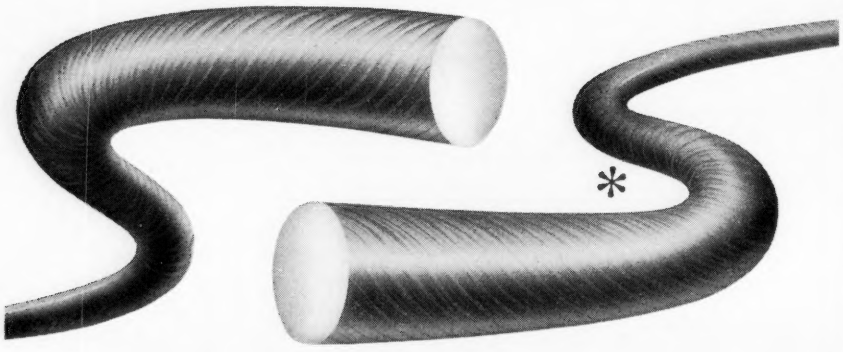

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The Club meets for luncheon on the first Thursday of each month except during the summer months. Meetings are informal, consisting primarily of discussions of administrative subjects proposed by the members. One dinner meeting for members and their wives is held each year.

The accompanying photograph includes all members of the club with the exception of GILBERT H. COLLINGS, JR., M.D., Standard Oil Company (Indiana); JERMYN F. MCCAHAN, M.D., Western Electric Company; JOHN TROXEL, M.D., Blue Cross-Blue Shield; and THEODORE R. VAN DELLEN, M.D., Chicago Tribune.

The present officers of the Medical Directors Club of Chicago are: President, N. GILLMOR LONG, M.D.; President-Elect, ROBERT M. GRAHAM, M.D.; and Secretary-Treasurer, JOHN POST, M.D.

Bottom row, left to right: Eugene E. Taylor, M.D., Hotpoint Company; Robert M. Watrous, M.D., Abbott Laboratories; Bryce B. Reeve, M.D., Standard Oil Company (Indiana); N. Gillmor Long, M.D., Lumbermens Mutual Casualty Company; Robert M. Graham, M.D., The Pullman Company; Arthur K. Peterson, M.D., R. R. Donnelly & Sons Company.

Middle row, left to right: Paul J. Whitaker, M.D., Allis Chalmers Mfg. Company; Andrew J. Oberlander, M.D., Prudential Insurance Company of America; Eugene L. Walsh, M.D., International Harvester Company; Allan P. Skoog, M.D., G. D. Searle and Company; George M. Roberts, M.D., Montgomery Ward and Company; Harold P. Lyon, M.D., Swift & Company; Bernard B. Michela, M.D., Rehabilitation Institute of Chicago; Richard J. Bennett, M.D., U. S. Steel Corporation; George H. Irwin, M.D., Chicago Transit Authority.

Top row, left to right: Edward H. Carleton, M.D., Inland Steel Company; Paul W. Rush, M.D., Corn Products Company; R. Vincent Kron, M.D., Campbell Soup Company; John Post, M.D., Zenith Radio Corporation; James N. Dewane, M.D., Libby, McNeill and Libby; John R. Winston, M.D., Santa Fe Railroad; John A. Palese, M.D., Liberty Mutual Insurance Company; James D. Thornton, M.D., Pennsylvania Railroad.

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REFERENCES:

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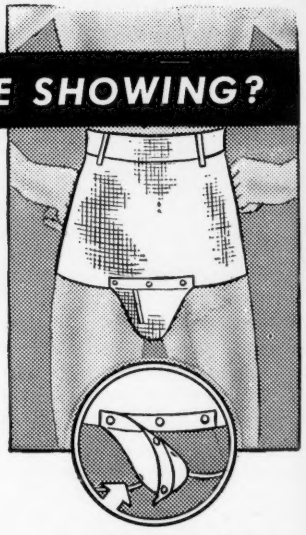

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